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# PEDIATRICS

## THE HYGIENIC & MEDICAL TREATMENT OF CHILDREN

BY

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VOLUME SECOND.

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the statements are merely approximate, but in this way the diagnosis of these diseases is much simplified and their characteristics are made more prominent.

TABLE 97.

	Variola.	Varicella.	Scarlet Fever.	Measles.	Rubella.
Incubation . .	12 days.	17 days.	4 days.	10 days.	21 days.
Prodromata . .	3 days.	A few hours.	2 days.	3 days.	A few hours.
Efflorescence .	Macules. Papules. Vesicles. Pustules.	Vesicles.	Erythema.	Papules.	Papules.
Desquamation .	Large crusts.	Small crusts.	Lamellar.	Furfuraceous.	. . . . .
Complications and sequelæ.	Larynx. Lungs.	. . . . .	Kidney. Ear. Heart.	Eye. Lung. Tuberculosis.	. . . . .

Although what I have shown you in this table is far from definite, and might, were one of the diseases to be diagnosticated, be very misleading, yet for differential diagnosis between all these diseases I think you will find it valuable because of its simplicity.

In addition to the leading points which I have indicated in the table, the general symptoms and the temperature of these diseases provide us with excellent material by which to distinguish one from the other.

The slow progressive development of variola is very distinct from the acute, rapid course of all the others. The vomiting and sore throat of scarlet fever are usually quite distinct from the coryza, lachrymation, and cough of measles. In variola the rise of temperature during the prodromal stage, its decided lessening at the time of the appearance of the efflorescence, and its gradual rise again during the stage of suppuration, are very distinct from the sudden rise of temperature in scarlet fever during the prodromal stage and up to the height of the efflorescence. In like manner the temperature in measles differs from that of the other diseases in its sudden rise on the first day of the prodromal stage, in its lessening on the second day, and in its rise on the third day and up to the height of the efflorescence. The manner of the decline of the temperature differs in variola, in scarlet fever, and in measles. While in variola it is slow and prolonged, in scarlet fever it is rather rapid, although it declines by lysis, and in measles the fall is often by crisis. In contradistinction to variola, scarlet fever, and measles, varicella and rubella differ markedly in the absence of a prodromal stage, in their short duration, and in their evanescent and moderate temperature.



## DIVISION XI.

### DISEASES OF THE NERVOUS SYSTEM, AND THE MYOPATHIES.

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#### LECTURE XXVII.

##### INTRODUCTION.

TO-DAY, gentlemen, we shall begin to study a class of diseases which is the most difficult to understand of any that are met with in early life. This difficulty exists necessarily from the complex organism of the parts affected, and on account of the important *rôle* which the nervous system plays in all diseases which occur in human beings during the process of their development. We are much more likely to meet with nervous phenomena of the most diverse varieties in children than in adults. In like manner we meet with the most widely differing clinical symptoms. If you had studied the clinical symptoms of nervous diseases in the adult only, you would be insufficiently prepared to diagnosticate properly from similar symptoms in the case of the child. Symptoms which if occurring in adults would be significant of serious lesions of the nervous system may arise in children from simple reflex conditions which only simulate and do not represent actual disease.

Children are much more apt to become unconscious, to have convulsive attacks, and to show disturbance of the functions of important nervous centres from reflex irritation, than are adults. The whole cerebro-spinal system in infancy and early childhood is so impressionable, so excitable, and so hypersensitive to even slight grades of irritation, that diseases of a nervous type, whether primary or secondary, dominate all others.

We have, then, not only well-recognized pathological lesions with their characteristic symptoms, as in adults, but also the same groups of symptoms caused by different pathological conditions, and, again, reflex nervous phenomena without organic lesions, *ad infinitum*.

These reflex phenomena are so much more numerous than those which arise from organic lesions, and are so irregular in their manifestations, that, from a diagnostic point of view, they are most important. They also enter



into all disturbances of the nervous system, whether functional or organic, to such a degree that what we have learned concerning cerebral localization in the adult becomes of much less value in the young subject. Attempts to locate minutely diseases of the nervous system by means of cerebral localization are so indefinite, and in the hands of the general practitioner so fruitful of incorrect conclusions, that I have thought it better to pay very little attention to this branch of neurology, which for the present should be referred to the nervous specialist.

Difficult as the study and clinical recognition of these manifold conditions are, far greater becomes our task when we attempt to classify and arrange in simple form for the purpose of teaching the complex nervous phenomena which we meet with in our nursery practice. Diseases of the nervous system constitute in themselves the study of a lifetime, and we who are busily engaged in general medicine cannot hope to obtain the exact detailed knowledge of the nervous specialist. The nervous specialist, on the contrary, who has not worked practically among children, studying them in all their various phases of excitement and rest, disease and health, may fail to grasp the special phase of nervous disease by which he is at times confronted.

The various pictures of nervous diseases which I shall present to you are those which you will be most likely to meet with in practice. As it is macroscopic rather than microscopic knowledge which is most needed for clinical observations in childhood, I shall treat the subject broadly, leaving the finer touches for your later and more extended study of the works of skilled neurologists.

We must adopt some division for teaching which by its simplicity will aid us to keep in mind the various diseases in a connected series. As the mind grasps more readily symptoms produced by a distinct pathological lesion than those where such a lesion has not been proved to exist, I shall speak first of the principal organic lesions of the brain and cord, reserving for later lectures what I have to say about the various nervous phenomena which from our indefinite knowledge concerning them we term *functional*. I have adopted this division simply for the purpose of clearness in teaching. It is not that of any especial authority on nervous diseases, but it is what I have found to satisfy practically the needs of the many classes of students whom I have met from year to year.

The terms anæmia and hyperæmia of the vessels of the brain, as designating distinct diseases, have been used frequently in connection with the discussion of diseases of the brain and cord. These terms should in the present state of our knowledge be restricted to represent symptoms, and not diseases, for in the majority of cases they are only symptoms which are secondary to some primary disease.

Nervous diseases can as a whole be divided, as may be seen in this table (Table 98, page 592), into—I. *Organic*; II. *Presumably Organic*; III. *Functional*.

TABLE 98.

I. ORGANIC . . . . .	Where there is a distinctly definite pathological condition.
Examples . . . . .	<i>Meningitis. Hydrocephalus.</i>
II. PRESUMABLY ORGANIC . . . . .	Where there is no definite lesion, so far as we can at present determine, but where we suppose that a pathological condition may in the future be discovered, and that the disease may then be relegated to the organic class.
Examples . . . . .	<i>Chorea. Epilepsy.</i>
III. FUNCTIONAL.	
1. Probably Central . . . . .	Where apparently the symptoms arise from a disturbance rather than a lesion of the nervous centres.
Examples . . . . .	<i>Hysteria. Temporary aphasia from fright.</i>
2. Reflex . . . . .	Where the symptoms are caused by peripheral irritation of various parts of the nervous system.
Examples . . . . .	<i>Convulsions from foreign bodies in the stomach. Asthma dyspepticum.</i>

Under each of these divisions I have tabulated the various diseases which belong to it, and I shall speak in detail only of those which you will be likely to meet with in general practice.

By referring to this second table (Table 99) you will see at a glance which diseases I am about to describe to you, and the order which I have followed in describing them.

I would also call attention to the fact that this table is not arranged on either a purely pathological or a purely symptomatic basis. On the contrary, wherever it seemed expedient to designate a disease by the name of its principal symptom I have done so, although in most cases I have used a pathological term. The table, then, does not represent a recognized scientific classification of nervous diseases, but is merely a list of the different diseases in the order in which I shall speak of them.

TABLE 99.

*Nervous Diseases.*

Nervous Diseases.			
I. Organic.		III. Functional.	
II. Presumably Organic.		Probably Central.	Reflex.
Non-tubercular meningitis.	Chorea.	Hysteria.	Pavor nocturnus (peripheral).
Tubercular meningitis.	Epilepsy.	Hypnotism.	Dental reflex.
Thrombosis of the cerebral sinuses.	Insanity.	Catalepsy.	Reflex nystagmus.
Hydrocephalus.		Simulated diseases.	Reflex of ear.
Cerebral abscess.		Insolation.	Reflex of larynx.
Cerebral paralysis.		Concussion.	Paroxysmal gasping.
Athetosis.		Temporary amnesia.	Reflex of lung.
		Temporary aphasia.	Reflex cough.
		Arrested psychical development.	Reflex of heart.



TABLE 99.—*Continued.**Nervous Diseases.*

I. <i>Organic.</i>		II. <i>Presumably Organic.</i>		III. <i>Functional.</i>	
				Probably Central.	Reflex.
Intra-cranial tumors.				Retarded speech.	Reflex of stomach.
Intra-cranial syphilis.				Headaches.	Reflex of bladder.
Idiocy.				Vertigo.	Reflex of vagina.
Mirror-writing.				Sensitive spine.	Reflex of rectum.
Myelitis.				Tetany.	
Poliomyelitis anterior.				Pavor nocturnus	
Paralysis from caries of the spine.				(central).	
Hereditary ataxia.					
Locomotor ataxia.					
Syringomyelia.					
Multiple cerebro-spi- nal sclerosis.					
Cerebro-spinal menin- gitis.					
Neuritis.					
Multiple neuritis.					
Paralysis of the new- born.					
Neuralgia.					

I must impress upon you the fact that the classification which we may deem best to adopt to-day will in all probability in the next five or ten years have to be modified by the further study of nervous pathology. You will notice, however, that in my classification I have allowed for this progress in medical thought, and that the various diseases which I have tabulated in the divisions "Presumably Organic" and "Functional" can be placed in the class of "Organic Diseases" as soon as it has been proved that they belong there.

In studying the various diseases of the nervous system which I shall explain to you, and the cases which illustrate them, I have received so much aid from the special knowledge of these diseases possessed by Dr. William N. Bullard that I wish to acknowledge my indebtedness to him.

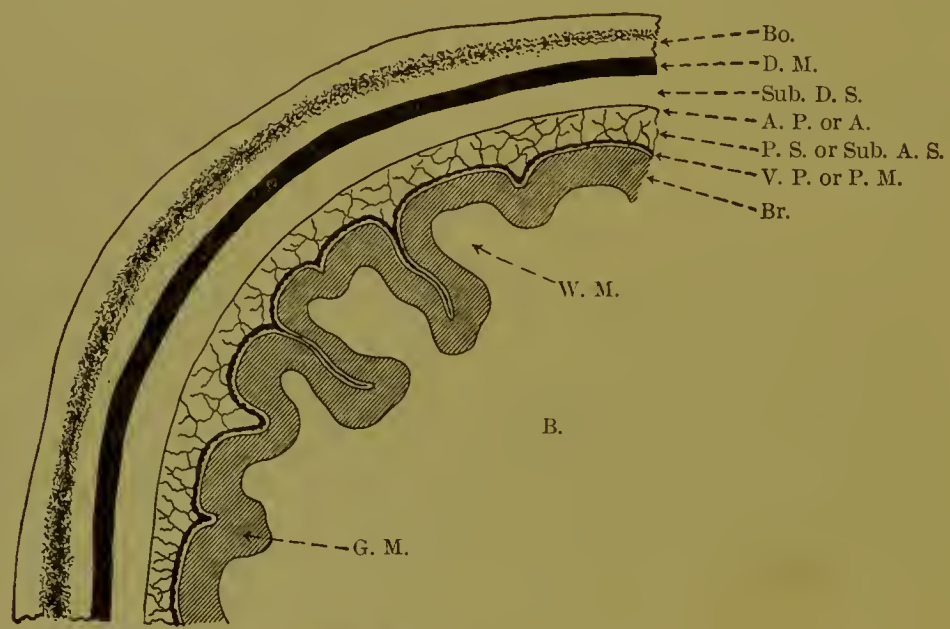
LECTURE XXVIII.

I. ORGANIC NERVOUS DISEASES.

BRAIN.—CORD.—BRAIN AND CORD.—PERIPHERAL NERVES.

**BRAIN.**—Before speaking of the diseases of the brain I should like to have you examine a section of the skull as shown in this diagram (Diagram 8). It represents the relations between the bone, the dura mater, the subdural space, the arachnoid, the subarachnoid space, the pia mater, and the brain. The diagram is useful for the clinical investigation of nervous diseases, and will, I think, aid you in understanding what I am about to describe.

DIAGRAM 8.



Section of skull and brain.

Bo. . . . .	Bone.
D. M. . . . .	Dura mater.
Sub. D. S. . . . .	Subdural space.
A. P. or A. . . . .	Arachno-pia or arachnoid.
P. S. or Sub. A. S. . . . .	Pial space or subarachnoid space.
V. P. or P. M. . . . .	Visceral pia or pia mater.
Br. . . . .	Brain.
W. M. . . . .	White matter.
B. . . . .	Brain.
G. M. . . . .	Gray matter.

The dura mater is closely attached to the skull at all ages, but especially so in childhood.

The subdural space lies between the dura mater and the arachnoid.

The subarachnoid space is crossed by fibres, thus making a connection between the arachnoid and the pia, which some anatomists are now inclined to speak of as one structure.



**MENINGITIS.**—If you will again glance at this table of classification (Table 99, page 592), and at the anatomical diagram (Diagram 8, page 594), you will see that I should naturally first speak of diseases of the cerebral meninges. Of these diseases *meningitis* is the most common. Cerebral meningitis may affect the *dura mater* or the *pia mater*. In the first case it is called *pachymeningitis*, and in the second *leptomeningitis*. The form may be *acute* or *chronic*.

**Pachymeningitis.**—Pachymeningitis is in early life so rare, except from certain local traumatisms, or as a lesion in some specific disease, such as syphilis, that we need merely mention it, and can at once proceed to study the inflammatory conditions of the *pia mater*.

**Leptomeningitis.**—Leptomeningitis, on the contrary, is very frequent in childhood. It may be divided primarily as to its *locality* into (1) meningitis of the convexities, and (2) meningitis of the base; as to its *pathology*, into (1) a simple non-tubercular inflammation of the *pia*, and (2) a growth of miliary tubercle in the meshes of the *pia* producing inflammation.

This is only a general division, but it serves to prepare you for the somewhat more minute description which is necessary to make you understand the varied clinical symptoms which are met with in these diseases, as the lines cannot be drawn sharply as to locality, pathology, or symptoms.

(1) **NON-TUBERCULAR MENINGITIS.**—Non-tubercular meningitis is often called *purulent meningitis*, but the latter term does not seem so applicable as the former, because we also meet with a purulent exudation in certain cases of tubercular meningitis. The pathological process may, although in a general way and to the greatest extent affecting the membranes of the convexity, attack the membranes of the central and basic regions of the brain. Following, however, the rule that where we are making a clinical division of diseases we should emphasize the salient lesions by which we can in most cases distinguish them, I shall leave the minute pathology of these diseases for your more extended pathological studies.

Non-tubercular meningitis in its acute form is a disease which may attack robust as well as debilitated children, and may occur at all ages. It is rare in the first year of life. It is most common in the middle period of childhood.

Clinically, we sometimes find the non-tubercular form in infants simulating in its symptoms, on account of the locality affected, the tubercular form of the older child. On the other hand, the tubercular form in infants is sometimes so acute in its symptoms as to simulate the non-tubercular form of the older child.

Some rare cases of non-tubercular meningitis have been reported where the disease was apparently primary, and for the present, therefore, we can speak of this class of cases as idiopathic until further light has been thrown upon the subject. I myself have never seen a case of non-tubercular meningitis which was undoubtedly idiopathic. The great majority of cases is secondary.

Non-tubercular meningitis is most frequently of traumatic origin, as from some injury to the head, or the disease may arise from disease in the ear with its local inflammation extending through the petro-squamosal suture to the cerebral meninges. A comparatively small number of cases of non-tubercular meningitis appears to be caused by the specific microbe of such diseases as scarlet fever, measles, erysipelas, pneumonia, possibly typhoid fever, rheumatism, and syphilis. It also occurs in cerebro-spinal meningitis, and in rare cases it is secondary to a group of symptoms to which the name insolation has been given.

This table (Table 100) designates the different causes which may give rise to non-tubercular meningitis.

TABLE 100.		
Non-tubercular Meningitis.		
Primary (said to exist).	Secondary.	
	Traumatic.	Ear.
		Specific diseases, such as
		Scarlet fever,
		Measles,
		Erysipelas,
		Pneumonia,
		Typhoid fever,
		Rheumatism,
		Syphilis,
		Cerebro-spinal meningitis,
		Insolation.

**PATHOLOGY.**—The pathology of non-tubercular meningitis is practically, where infants and young children are concerned, an inflammation of the pia mater. This, according to Delafield and Prudden, may be acute, chronic, tubercular, or syphilitic.

In any case of acute meningitis the inflammation is apt to extend downward and to involve the pia mater of the cord. In young children it especially happens that the inflammation may involve the ependyma of the ventricles and cause a distention of these cavities with serum.

In one form of acute non-tubercular meningitis the pia mater, according to Delafield and Prudden, from whose observations I shall freely quote, is somewhat congested. Its surface is dry, lustreless, and somewhat opaque. These changes in the gross appearances of the membrane are not marked, and may be overlooked, but the minute changes are more decided.

There is an abundant production of cells somewhat resembling the cells which coat the surfaces of the membranes and fibres which make up the pia mater. The cell growth is general, involving the pia mater over most of the surface of the brain. The inflammation is one which results in the production not of fibrin, serum, or pus, but of new connective-tissue cells. This form of meningitis, which may be called *acute cellular meningitis*, is of



frequent occurrence, and is attended with the ordinary clinical symptoms of acute meningitis.

Another form of acute non-tubercular meningitis has been termed the *exudative*, because it is characterized by the accumulation, chiefly in the meshes of the pia mater and along the walls of the blood-vessels, of variable quantities of serum, fibrin, and pus. Sometimes one, sometimes another, of these exudations preponderates, giving rise to serous, fibrinous, or purulent forms of inflammation. The absolute quantity of the exudation varies greatly. In some cases death may be caused with so slight a formation of exudation that to the naked eye the pia mater may look quite normal or, perhaps, only moderately hyperæmic or œdematous. The microscope, however, in these cases will reveal pus-cells in small numbers, and sometimes flakes of fibrin in the meshes and along the walls of the vessels. In other cases turbid serum in the meshes of the membrane is all that can be seen, and the turbidity is shown to be due to pus-cells or to a small amount of fibrin. Again, either with or without marked œdema of the pia mater, yellowish stripes are seen along the sides of the veins, sometimes appearing like faint turbid streaks, and at other times dense, opaque, thick, and wide, so as almost to conceal the vessels. These are due to the accumulation of pus-cells and fibrin in large quantities along the vessels. They can be seen best and are most abundant around the larger veins which run along over the sulci. In still other cases the infiltration with pus and fibrin is so dense and thick and general that the brain-tissue, the convolutions, and most of the vessels of the pia mater are concealed by it. This is usually of a greenish-yellow color, and is sometimes so thick as to appear like a cast of the brain-surface at the seat of the lesion. Sometimes extravasated red blood-cells are mingled with the other exudations as the result of diapedesis. Microscopic examination shows numerous white blood-cells sticking in the walls of the veins and capillaries, or the vessels may be blocked with them. It is evident that a large part of the pus-cells accumulates as the result of emigration. The connective-tissue cells of the pia mater may be detached from their places or degenerated. In some cases there are considerable accumulations of pus between the pia mater and the brain-substance and along the vessels which enter the latter. More rarely, pus is found upon the free surface of the membrane. The brain-substance may be compressed by the accumulated exudation so that the convolutions are flattened. The cortical portion of the brain may be simply infiltrated with serum (œdematous), or it may undergo degenerative changes and may be the seat of punctate hemorrhages. Not infrequently the inflammation extends to the ventricles, which may contain purulent serum, and to the pia mater of the cord. This form of infiltration is most frequent on the convexity of the brain, but may extend or even be confined to the base. It may be localized, but it frequently extends widely over the surfaces of the hemispheres. Bacteria are often present in the exudation, and I shall explain their relationship to the lesions when speaking of cerebro-spinal meningitis.

When recovery occurs from the *acute exudative form* of non-tubercular meningitis, there may be fatty degeneration of the cells which have accumulated in the pia mater, particularly along the vessels, and this may produce white patches in the membrane and threads along the blood-vessels, which resemble the accumulation of exudation in the acute stage. Fatty degeneration of the blood-vessels and cells of the pia mater may also occur without acute inflammatory changes. Sometimes in children inflammatory changes in the ventricles persist for days and weeks after the subsidence of the inflammation of the pia mater.

The non-tubercular form of meningitis may also be chronic, in which case the pia mater at the base of the brain alone may be inflamed (basilar meningitis), or only the pia mater over the convexity, or the entire pia mater, or certain circumscribed patches of the membrane. In these cases the pia mater is thick and opaque, and there is a formation of new connective tissue, with a production of pus, fibrin, and serum. The relative quantity of these inflammatory products varies in different cases, and results in some cases in firm and at times extensive adhesions between the dura mater and the pia mater. Other conditions which represent the results of chronic inflammation may also be present, but need hardly be referred to here, further than to say that the ventricles of the brain may in this chronic form contain an increased amount of serum and may be dilated. The ependyma also may be thickened and roughened.

**SYMPTOMS.**—Where *non-tubercular meningitis* is secondary to injuries or to other diseases, the characteristic symptoms may of course be complicated and even obscured by symptoms resulting from the especial cause. In the supposed primary or idiopathic cases the symptoms, especially where the child is over two years of age, are rapid in their development. The course is short, from seven to eight days, and the disease may often prove fatal in forty-eight hours. The disease, when affecting the convexities chiefly, begins with intense headache and a high temperature,  $40^{\circ}$ – $40.6^{\circ}$ – $41.1^{\circ}$  C. ( $104^{\circ}$ – $105^{\circ}$ – $106^{\circ}$  F.). The respirations are rapid, 30–40–50, and comparatively regular. The pulse is quick, 150–160–170, but is usually regular. Vomiting, photophobia, contracted pupils, and delirium are present. Convulsions occur early. Later we may have blindness and paralysis.

**DIAGNOSIS.**—As the diagnosis of non-tubercular meningitis is chiefly a differential one from tubercular meningitis, I shall reserve what remains to be said on this subject until I speak of the latter disease.

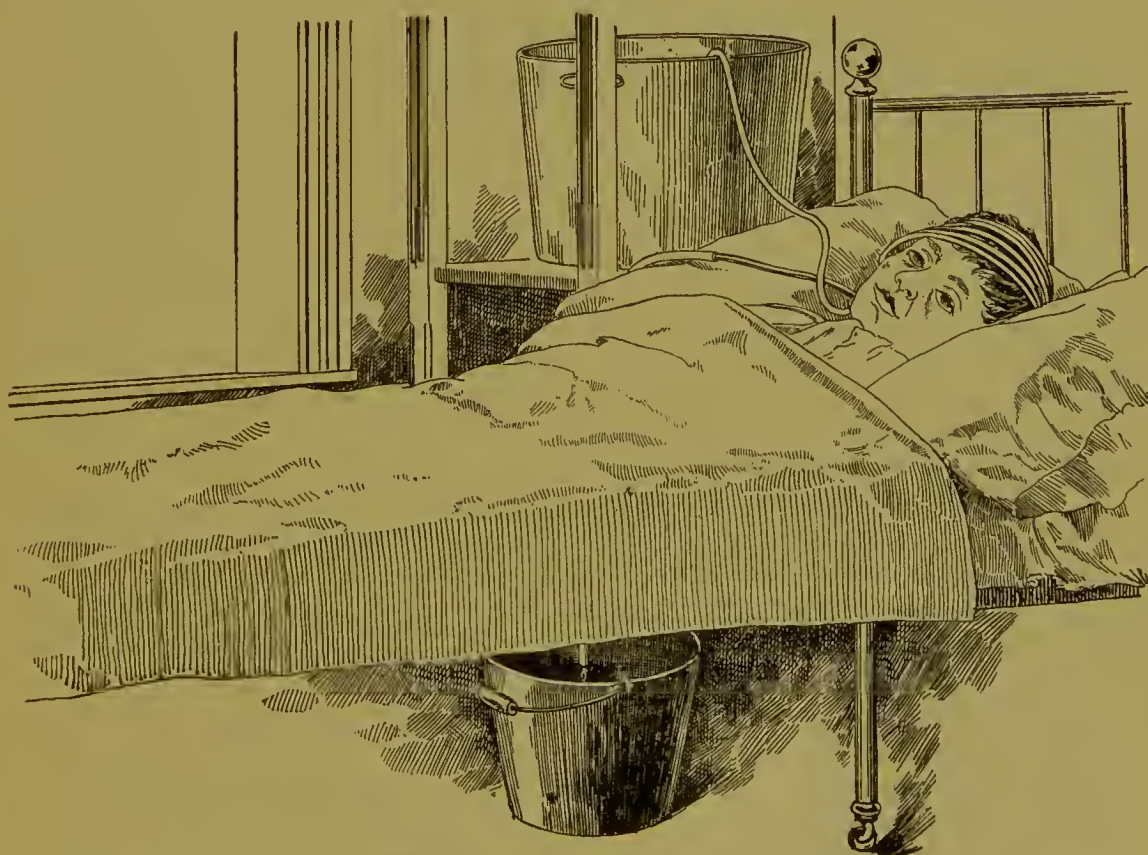
**PROGNOSIS.**—The prognosis is very unfavorable. It is possible, however, for the child to recover completely from an attack of non-tubercular meningitis. Perhaps only a changed mental condition will remain, boys appearing effeminate or more easily excited than would be considered normal. Some of the more acute forms affect also the brain, and we find their results in idiocy and contractures. We must always bear in mind that children have wonderful recuperative powers. Their nervous organisms, although sensitive to the least shock or the slightest irritation, from



the activity of their growth present opportunities for repair which do not occur in adults. So long as a disease of a necessarily fatal character is not present, the possibility of recovery should not be lost sight of. Violence of the nervous manifestations does not by any means always indicate a fatal issue.

**TREATMENT.**—The treatment of non-tubercular meningitis varies with that of the disease or condition to which it is secondary. The child should be kept in a cool, dark room and protected from noise. In the treatment of meningitis, whether it is a symptom or whether it is idiopathic, the indications are to reduce the temperature of the body and to support the general strength until the disease has run its course. The former is accomplished best by the application of mustard derivatives to the lower extremities, by sponging the entire body every three or four hours with water at a temperature of from  $15.55^{\circ}$ – $22.22^{\circ}$  C. ( $60^{\circ}$ – $70^{\circ}$  F.), and by the application of cold to the head. The strength should be supported by the administration of milk, and, when necessary, of stimulants.

## CASE 258.



Treatment of meningitis with Leiter's coil.

The method of applying cold to the head by means of Leiter's coil is a valuable one, and I have here in this bed a child (Case 258) with meningitis who is being treated in this way.

The apparatus called Leiter's coil is very simple, and consists of a light flexible metallic or preferably rubber tubing, which can be bent in any way desired and applied to any part of the body or limbs as well as to the head.



Two vessels are needed : one at a height somewhat above that of the child's head acts as a reservoir for the water, while the other stands under the bed to receive the water after it has passed through the tube. In this way we can have water at a constant temperature, warm or cold, continually running through the tube several times around the child's head.

In addition to the local treatment, bromide of soda in varying doses, according to the age of the child and the severity of the disease, can be given.

Dr. Fraser reports the case (Case 259) of a male infant, fourteen months old, unusually well developed and previously perfectly well. It began to be irritable and to lose in weight. These symptoms continued for about a month. When it was brought to him it had a temperature of 37.4° C. (99.5° F.), and it had no other symptoms beyond what would be expected from the condition of the gums, which were hot and tender. Three days later a convulsion occurred, and two days later hemiplegia of the left side. The pulse was 130, small and irregular. The temperature was 38.6° C. (101.5° F.). Sensation was perfect on both sides. On the following night the infant began to have convulsions, which continued with irregular intervals until the next morning. The entire voluntary muscular system was then found to be in a state of tonic spasm. The legs were rigid, the head was retracted on the trunk, and there was opisthotonos. This tonic spasm was interrupted at intervals of half an hour by a clonic seizure involving chiefly the extremities. While these nervous manifestations continued, the thumbs and the fingers were bent into the palms, and the forearms were flexed and extended upon the arms with short rhythmical movements.

The inferior extremities were similarly affected, though to a milder degree. The movements also extended to the face, giving rise to contortions. The respiration was irregular, but there was no lividity of the skin. The pulse was 140. The temperature was 38.8° C. (102° F.). Three days later, the previous symptoms having in the mean time continued, there was a diminution in the convulsions, but consciousness almost entirely disappeared, and there was an increasing tendency to coma. The pupils were contracted, there was an entire inability to swallow, and the infant gradually sank, dying at 6 P.M.

The post-mortem examination was made twenty-four hours after death. On opening the skull and deflecting the dura mater the convolutions appeared flattened, as if they had been slightly compressed. The veins of the cerebral cortex were much engorged. The outer surface of the visceral layer of the arachnoid was smooth and dry, but at a spot about 1.2 cm. ( $\frac{1}{2}$  inch) in diameter, situated about the middle of the ascending frontal and parietal convolutions of the left hemisphere, the pia mater was covered by a thin, yellowish layer of lymph. During the removal of the brain several ounces of clear serous fluid escaped from the lateral ventricles. On section of the hemispheres the centrum semi-ovale did not present any unusual number of vascular points on either side, but the substance of both hemispheres, especially that of the left, was very soft.

The optic thalamus and lenticular nucleus of the left hemisphere were so much softened as to be almost diffuent. The ependyma of the lateral ventricles was soft and uneven, and it appeared in parts to be covered by a layer of lymph, but the surrounding tissues were so much softened that it was doubtful whether the layer consisted of lymph or of the smooth and softened ependyma. At the base of the brain a layer of lymph 0.3 cm. ( $\frac{1}{8}$  inch) in thickness was found in the interpeduncular space underneath the visceral layer of the arachnoid. The inner surface of the dura mater at the base of the skull was smooth and without a trace of opacity.

There was in this case a softening of the brain-substance, which was probably secondary to the meningitis.

This boy (Case 260), four years old, whom I have here to show you, is apparently suffering from the results of non-tubercular meningitis. He was always well and strong until the onset of the present attack, which occurred twelve weeks ago. He never has had any disease, with the exception of measles when he was three years old.

This last attack, in all probability, was produced by a fall, in which he struck the back of his head. No cut or bruise was detected. Later, on the day of this fall, he began to complain of pain in his head and to vomit. He was very feverish, and lay in bed protecting his eyes from the light, as there was great photophobia. The bowels were regular, and he took small quantities of food. A week later he became delirious, and this condition continued for two weeks. He was then brought to the Children's Hospital, and from time to time was delirious during a period of five weeks. The delirium was sometimes active, and then it would disappear and he would recognize his parents. He was very cross in the intervals of the delirium, and would roll his head from side to side. His appetite was poor. He never had any convulsions or paralysis. For a time, however, he had incontinence of urine.

Since this attack he has been gradually growing better, and he is now comparatively well, although he sometimes complains of slight pain in his head, at which time the head feels hotter than at others. He also sometimes has a little photophobia, and when exposed to unusual heat or excitement is rather restless and fractious. His pupils have seemed to be slightly dilated, but their reaction is normal.

The treatment has been simply to keep him perfectly quiet. His diet has been carefully regulated, and 0.18 gramme (3 grains) of bromide of potash has been administered several times during the day. At present his pulse is 98 and regular, his temperature is 36.2° C. (92.7° F.), and his respirations 25 and rhythmical.

The diagnosis is probably traumatic non-tubercular meningitis.

The next case (Case 261), which I have had brought to show you as possibly one of non-tubercular meningitis, is a child two and one-half years old.

He was healthy at birth, and remained so until he was eleven months old, when he had an illness lasting for two or three weeks, characterized by high temperature, but no other definite symptoms beyond apparent irritation connected with the teeth.

When he was seventeen months old he had a similar attack, only more severe, accompanied by delirium, photophobia, high temperature, and, in a few days, paralysis of the legs and left arm, while he could only move the right arm slowly. He had a tendency to turn the head to the right, and his head was retracted. The faecal movements and the urine were normal. He was unconscious for two days. An examination showed that there was nothing abnormal in the ears, nor was anything abnormal found on physical examination elsewhere. He cried out as though he had severe pain in his head when the attack began. After a few days he began to improve rapidly, and, although he had never talked before, soon began to express himself in words.

During the following year he had some trouble with his ears, and grew very weak, so that he could not walk. Later he had an attack of croup, accompanied by perforation of both membranæ tympani.

To-day, as you see, he is comparatively well.

In regard to the diagnosis of these last two cases, we are only justified in saying that if they continue well, and do not show a return of cerebral symptoms, the most probable explanation of their condition is a non-tubercular meningitis.

In this next bed is a little girl (Case 262), four years old, who fell and struck the back of her head. She did not complain of much pain until the next day, when in the evening her face was flushed, she vomited, was restless, and was constipated. On the next day the symptoms increased in severity, and two days later she entered the hospital. Her head was retracted. There was an erythematous condition of the skin of the face, elbows, and knees. The pupils were equal and reacted well. She was very restless, but showed no evidence of pain. Her pulse was 132, the respirations were 44, and the temperature was 39.1° C. (102.4° F.). She was able to take nourishment and to retain it. She had marked opisthotonos. During the following night and day she moved her hands continuously, and early in the morning vomited. The erythema of the skin gradually faded away. She talked incoherently most of the time. The eyes were fixed. The feet and hands were cold.

Yesterday at times she showed labored breathing and the pupils were dilated. The head was not retracted so much, but the muscles of the neck were very stiff. The pulse was much more feeble and slower. The abdomen was retracted, and there were petechiæ

on the face, elbows, and knees, most marked on the right side. Last night she was very restless and her breathing was again labored.

To-day, as you see, there is considerable twitching of her arms and legs. The patellar reflexes are absent; the plantar reflexes are diminished. Nothing abnormal is found on examination of the ears, throat, chest, and abdomen, or of the urine.

This case is probably one of traumatic non-tubercular meningitis. The erythematous efflorescence and the petechiæ would make me suspect that we might possibly be dealing with a case of cerebro-spinal meningitis. The symptoms, however, are not of so severe a grade as I should expect in the latter disease, while the acute onset following trauma would naturally point towards a simple inflammation of the pia mater. We cannot, however, in cases of this kind definitely determine the diagnosis without an autopsy. It is evident that there are no other diseases, such as typhoid fever or pneumonia, developing, and the possibility of its being a tubercular meningitis is exceedingly small, considering that she is at a period of childhood when the typical signs of this disease are most marked, and its type is of a subacute character.

(Subsequent history.) On the following day she sank rapidly, and she died, without any spasmodic movements or convulsions, on the eighth day of the disease.



## LECTURE XXIX.

## BRAIN.—(Continued.)

## TUBERCULAR MENINGITIS.

THE second form of leptomeningitis which I shall describe to you is called tubercular meningitis, and I happen to have a number of children illustrating this disease in the wards of the Children's Hospital to show you to-day.

Tubercular meningitis is a disease caused by the tubercle-bacillus attacking the pia mater; it occurs most commonly in early life, runs a sub-acute course, and is invariably fatal. The disease presents many irregularities in its manifestations, and its typical symptoms vary according to the age of the patient. The most typical cases of the disease are seen in middle childhood. It occurs more commonly between the ages of five and seven than at any other period of life. It is rare in the first year of life, especially in the early months; the number of cases increases rapidly in the second year and decreases as rapidly after the eighth year. It is comparatively so rare in adult life that out of the large number of adult patients that I have met in my service at the City Hospital only a few cases of tubercular meningitis have come under my care in the last ten years. Tubercular meningitis, then, can be considered to be essentially a disease of early life, and to be most common in the middle period of childhood. In a large number of cases there is a tubercular history of one or both parents. It is hereditary in the sense that the individual inherits tissues which are more or less receptive to and which provide a favorable material for the development of the bacillus of tubercle.

Every child should be protected in all possible ways against tubercular infection, whether by its food or by human beings. The tubercle-bacillus appears at times to attack individuals in cases where the question of inheritance can absolutely be eliminated. We should, therefore, take the greatest care that children should not be under the care of tuberculous nurses, as the nurse is the member of the family who comes into the closest relation with the child. As an illustration of the truth of this statement I shall mention a case seen by me in consultation with Dr. W. L. Richardson and Dr. H. P. Jaques.

A boy (Case 263), five years old, died of tubercular meningitis. The autopsy showed extensive tubercular lesions of the meninges, with enlarged bronchial lymph-glands and cheesy nodules at the apices of both lungs. The child up to the time of the attack had always been perfectly well. There was no history of tuberculosis on either the father's or the mother's side. There were several other children in the family, none of whom had ever

shown any symptoms connected with tuberculosis. This boy at the age of fourteen months was placed in the charge of a nurse about twenty years old, who remained with him until he was four and a half years old. Just before leaving the child she was brought into especially close connection with him while his parents were away for some weeks. The child was very fond of his nurse, insisted upon being in her lap a great deal, kissed her on the mouth, slept in her bed, and kept her in the nursery with him continuously. This nurse had a sister who died of pulmonary tuberculosis. She herself was taken sick with the same disease while taking care of the child, and subsequently died of it.

Other cases of this kind have been known to occur. Of course the possibility of a coincidence must be thought of, but the fact that a robust child with no hereditary tuberculous history lives in close connection with a tuberculous nurse and dies of tuberculosis of the bronchial glands and cerebral meninges is at least significant.

It is not unusual to meet with a tubercular meningitis secondary to tubercular disease of the spine. This complication occurred in a child four years old whom I saw in consultation with Dr. Seudder.

The child (Case 264) was being treated for Pott's disease with lateral deviation of the spine. He was placed on a frame for five weeks, and at the end of that time he lost in appetite and weight and began to have a cough. Nothing especial, however, was found in the lungs. The bowels became constipated, and he then began to have some mental disturbance and to vomit. A few days later he became unconscious, and on examining him his pupils were found widely dilated, uneven, and not responding to light. His temperature was usually about  $38.8^{\circ}$  C. ( $102^{\circ}$  F.). The pulse and respirations were somewhat quickened. His head was retracted, and on the day of his death he had a convulsion.

Tubercular meningitis may also occur in connection with disease of the hip, the latter being much more common than when the spine is affected. I have seen a case of this kind in consultation with Dr. Brown which illustrates the importance of recognizing the occurrence of this complication.

A child (Case 265), four years old, was being treated by an irregular practitioner for disease of the hip-joint. The child had been allowed to drag itself about, and the treatment had been with drugs and not by apparatus. When the child was placed under Dr. Brown's care he had him taken to the country and placed in a house and room where all the hygienic surroundings were good. He kept the child in bed and treated it by means of the method of extension usually employed in these cases. The child at first began to improve, but after a few weeks lost in weight and in appetite. Its temperature, which had been varying from  $37.2^{\circ}$  to  $37.7^{\circ}$  C. ( $99^{\circ}$  to  $100^{\circ}$  F.), rose to from  $39.4^{\circ}$  to  $40^{\circ}$  C. ( $103^{\circ}$  to  $104^{\circ}$  F.). A few days later the child became somnolent and had convulsions.

When I saw the case with Dr. Brown it was evidently one of tubercular meningitis, apparently secondary to disease of the hip-joint, and the child died within twenty-four hours after I had examined it.

In this connection I might mention that the tubercular form of otitis is not uncommon, and that it may be the starting-point for tubercular meningitis. Surgeons should, therefore, watch carefully the possible complication of tubercular meningitis when treating tuberculous disease of the bones and joints.

A knowledge of the general pathology of tubercular meningitis is of great practical importance in acquiring a clear picture of the disease. We must look upon the tubercular lesions as secondary manifestations of a



primary infection by the tubercle-bacillus of some other portion of the body, such as the bronchial or the mesenteric glands. Tubercular meningitis, therefore, is merely a part of a general tuberculosis. It, however, in early life is so prominent a part of tuberculosis, both in its clinical symptoms and in its pathological lesions, that I have placed it, not, as is usual in adults, under the heading of a general tuberculosis, but as a separate disease in my division of diseases of the nervous system.

**PATHOLOGY.**—Although the nidus of the tubercle-bacillus which produces the pathological lesions of tubercular meningitis is in some other part of the body, and the lesions of the brain and its meninges are always secondary, yet, as the clinical characteristics of the disease are those of a primary cerebral nature, I shall describe only the morbid lesions which occur in the brain.

The macroscopic pathological condition which is seen in the brain as a result of the action of the tubercle-bacillus is a growth of miliary tubercle in the meninges and in the cerebral substance. This growth is especially marked in the meshes of the pia mater along the course of the blood-vessels at the base of the brain. These small granulations are conspicuously numerous in the choroid plexus and cause great irritation in the neighboring parts. This irritation is followed by a transudation of greater or less extent into the ventricles. Accompanying this transudation is also a fibrino-purulent exudation between the pia mater and the cerebral convolutions at the base of the brain, notably in the fissures of Sylvius, but at times covering the whole convexity of the brain. The amount of exudation is not proportionate to the number of tubercles. The ventricles are sometimes so distended as to burst the septum. Pressure is thus brought upon the central portions of the brain, involving especially the optic thalamus, the corpus striatum, and the corpus callosum. While, as I have stated, the symptoms vary in different individuals and at different ages, the pathological lesions, on the other hand, with the exception of their location, are comparatively stable. What is of especial interest to us clinically, however, is that, although in a typical case of tubercular meningitis in middle childhood the symptoms, as a rule, correspond to the pathological lesions, yet in some cases we find an entire lack of such symptoms as would naturally result from the wide-spread and prominent lesions.

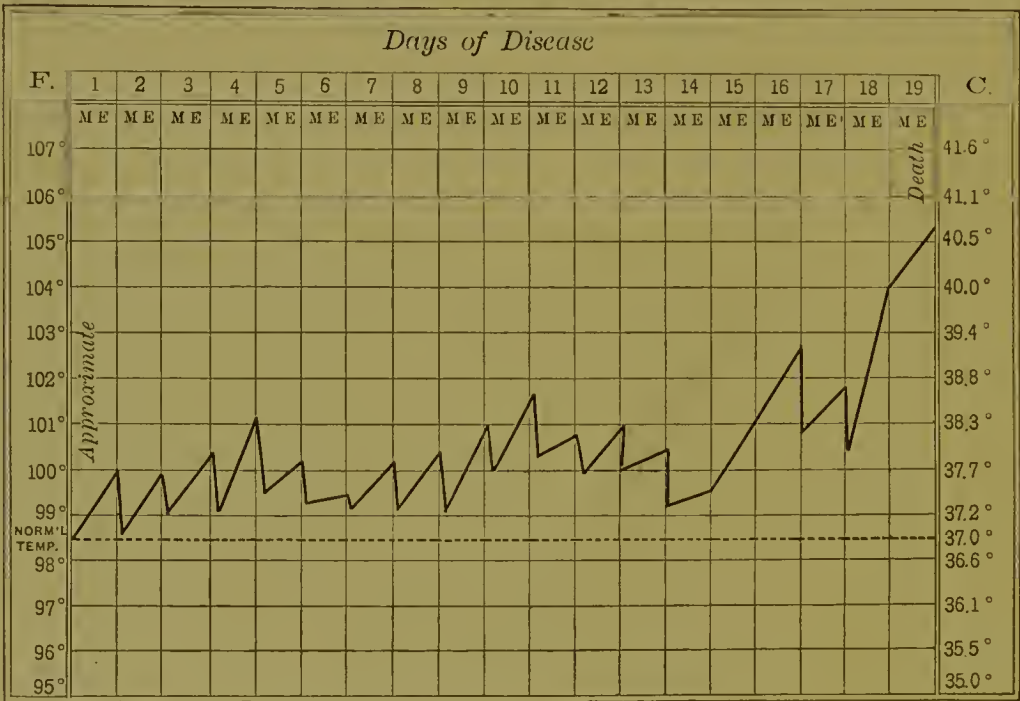
**SYMPTOMS.**—From what I have already told you in describing the symptoms of tubercular meningitis, we should first consider the course and the typical symptoms of the disease as it occurs in the middle period of childhood, and then state the variations which occur in infants.

By carefully studying the pathology of tubercular meningitis we can almost deduce the sequence of symptoms which we should expect to meet with in the middle period of childhood. In fact, in the great majority of cases occurring between the ages of two and eight years this sequence is very striking. Remember that as we are dealing with a symptom of general tuberculosis we should expect to find in the early stages of the disease that



the nutrition is affected, that there are a lessened appetite, loss in weight, anaemia, and in fact symptoms which warn us that something is affecting the child's general health. This condition may last for many weeks, or even months, varying as to the time when the tubercle-bacillus has left its original nidus and migrated to the cerebral meninges. Only after this has occurred do we begin to get symptoms of cerebral irritation. The child now becomes peevish and capricious, and is in some cases easily frightened. As the tubercular growth increases and causes further congestion of the blood-vessels, the sleep is disturbed; the child complains of dizziness and slight evanescent pains in the head; it staggers slightly in its walk (static ataxia); sometimes it cries out sharply, especially at night (hydrocephalic cry). Vomiting not apparently connected with the food, and usually without nausea, is a common symptom. These are symptoms of irritation of the nervous centres, and may last for a week or two, according to the development of the pathological lesions. The temperature is usually moderately raised, 37.2°–37.7°–38.3° C. (99°–100°–101° F.), but on some days it rises a degree or so higher, and just before death a considerable elevation may occur. This chart (Chart 19) shows the temperature of a

CHART 19.



Tubercular meningitis. Male, 4 years old.

child five years old who lately died in the hospital. It represents very well what you will usually meet with in uncomplicated cases of tubercular meningitis. Of course it is impossible to determine the exact day of the beginning of the disease in such an affection as tubercular meningitis, so that the first day marked on the chart is merely approximate and serves as a starting-point to show the character of the temperature.

The pulse at first is somewhat quickened, but it soon becomes slower than

normal, and is apt to intermit. The respirations may in the early part of the disease be quickened, and at times are of a sighing character. Obstinate constipation is a common symptom. Hyperæsthesia of the skin, with occasional waves of congestion, especially of the cheeks, is sometimes met with. The pain in the head increases, and the child is apt to hold its hand to its head. Drowsiness, at first slight, soon becomes very marked. The child is apathetic and lies in bed, refusing to eat. The urine is scanty. There is photophobia, and the pupils are contracted. Tubercles in the fundus of the eye are rarely seen during life. Abdominal pains are quite frequent, and depression of the abdomen (boat-shaped) is noticed in a certain number of cases. Drawing the finger over the skin usually produces a bright red line, which becomes in a few minutes quite intense, and lasts perhaps ten or fifteen minutes, which is much longer than would be the case in a healthy child. This phenomenon is called the *tache cérébrale*, and is quite frequently met with in tubercular meningitis, though it may be absent. This sign is, however, in no sense typical, and is seen in a number of other diseases. The child at this stage of the disease is apt to roll its head on the pillow almost continuously.

The pathological irritation has now gone on to exudation, and we begin to get symptoms of pressure. If diarrhœa appears, we should suspect tubercle of the intestine. Sopor now comes on rapidly, and the child can be aroused only at times. Strabismus, nystagmus, and ptosis may appear. The pupils are dilated and irregular, and their reaction is lost. The Meibomian secretion is sometimes markedly increased. Convulsions, generally partial, and of a rather mild type, may appear. At times paralysis of the arm, or of the arm and leg (hemiplegia), and interference with sight (optic neuritis), may occur. In some cases the pulse now becomes markedly slow and irregular, 50–60–70, and it is very common to find an intermission in the

CHART 20



Pause.

Cheyne-Stokes respiration. Tubercular meningitis. Child, 4 years old.

pulse, though this must not be considered as diagnostic of tubercular meningitis. The respirations may not be perceptibly diminished at first, but soon become slow, 10 to 15 in a minute. A peculiar form of respiration, called Cheyne-Stokes, usually occurs at this stage of the disease. This type of respiration is characterized by complete or almost complete cessation of the respiratory movements for a number of seconds. This is followed by a faint return of the respiratory movements, which gradually increase in depth, rising for five or six inspirations and then fading away again so as to be imperceptible. This chart (Chart 20) represents this type of respirations

occurring in the third week of the illness of a child four years old who died of tubercular meningitis.

A heightened temperature in tubercular meningitis indicates a complication of some kind, such as pulmonary tuberculosis, pneumonia, or tubercle in the intestine. At the end of the disease, however, the temperature rises rapidly, as do the pulse and respirations. Hearing, taste, and smell seem to be unimpaired for some time. The position which children with tubercular meningitis often take is somewhat characteristic. In all forms of meningitis they are apt to bury their heads in the bedclothes. There is often spasmodic retraction of the head, and they are inclined to lie with their knees drawn up. There may be spasmodic opisthotonos, as in this case which I shall presently show you (Case 272, page 618). The disease varies in its length, but usually lasts for from three to six weeks. Death may be preceded by continued convulsions for perhaps several hours. A striking feature which not infrequently occurs in the course of these general symptoms is a partial return to consciousness after the child has been lying in a stupor for several days. This phenomenon often induces the parents, and sometimes even the physician, to entertain hopes of improvement. It is, however, always delusive, for it has no favorable significance, and is soon followed by a more profound state of unconsciousness. These symptoms which I have mentioned do not, of course, always appear together, but may be present in different groups, varying with the individual. All the symptoms may disappear temporarily. There may be tonic as well as clonic contractions of the limbs and rigidity of the neck.

**DIAGNOSIS.**—The diagnosis of tubercular meningitis in the middle period of childhood, and with the sequence of symptoms which I have just enumerated, is not difficult, but you will at once perceive that the diagnosis in the early days or even in the first week of the disease must necessarily be very difficult. It is by watching the course of the symptoms and their general grouping, rather than by the consideration of any one symptom, or even one group of symptoms, that we are justified in making a definite diagnosis. The diagnosis, then, must, as a rule, be held in abeyance for many days. Reflex vomiting, with a moderate temperature, irregularity and intermission of the pulse, apathy, and many other symptoms of tubercular meningitis, I have often seen, both alone and in combination, in cases where they represented no cerebral lesion whatever. The active development and sensitive condition of the nervous system in childhood are so exaggerated in comparison with those of adults, that whatever disease may be present is liable to produce so profound an impression on the child's nervous centres that actual disease of these centres is readily simulated. Thus for days these apparently cerebral symptoms may mask by their undue prominence the symptoms of the real disease.

Illustrative of this difficulty are certain cases (Cases 466, 467) of pneumonia, which I shall describe to you in a later lecture (Lecture XLIX., page 984), in which the children had constant vomiting, soon became apa-



thetic, and later were unconscious. They rolled their heads, had a medium temperature and an irregular pulse, and one of them showed irregularity of respiration. These symptoms lasted for five or six days, and disappeared with the development of an apex-pneumonia.

**DIFFERENTIAL DIAGNOSIS.**—The differential diagnosis must be made between meningitis in general and other diseases, such as (1) *diseases of the stomach*, (2) *poliomyelitis anterior*, (3) *pneumonia*, (4) *malaria*, (5) *typhoid fever*, (6) *syphilis*, (7) *rheumatism*, (8) *nephritis*, (9) *cerebro-spinal meningitis*, and (10) *non-tubercular meningitis*.

(1) **Diseases of the Stomach.**—Unless the child is very young, acute gastric symptoms are, as a rule, not difficult to recognize after the first few days. We may at times, however, be suspicious of cerebral disease on meeting in an infant with continual vomiting and an elevated temperature where there is no discoverable source of reflex irritation to account for the symptoms. This is especially the case if there are some irregularity of respiration and a slow pulse. These may be cases of tubercular meningitis such as I have described that disease in the first year of life. Again, however, they may be simply cases of reflex vomiting. As illustrative of this class of reflex gastric disturbance I shall cite this case :

A male infant (Case 266), eight months old, was attacked with vomiting which lasted with short intervals for two days. There were apathy and slow, intermittent pulse. The temperature was 37.2° C. (99° F.). There were irregular respiration and rapid emaciation. This patient made a perfect recovery in four or five days, and the case was evidently of gastric origin. The slow, intermittent pulse, and the moderate temperature, which would have been so alarming in an older child, led me in this case, as in others in the first year of life, to eliminate tubercular meningitis. In my experience this interpretation of symptoms has proved to be correct.

(2) **Poliomyelitis Anterior.**—The following case of poliomyelitis anterior resembled tubercular meningitis: it is, however, the only one which I have seen where the resemblance of the two diseases was so striking :

A boy (Case 267), eighteen months old, showed for over a week symptoms closely simulating those of tubercular meningitis. Obstinate constipation and apathy were present, followed by unconsciousness; there were also a marked *tache cérébrale*, distended fontanelle, irregular pulse, contracted pupils, eyes turned upward, and convulsive attacks. Finally, paralysis of one of the arms appeared, the general symptoms passed off, and the diagnosis of poliomyelitis anterior was easily made.

(3) **Pneumonia.**—The cases (Cases 466, 467) of pneumonia which I have referred to warn us that we should hold our diagnosis in abeyance, sometimes even for a week.

(4) **Malaria.**—Although we must admit that malaria closely simulates almost any disease, it is not usual to mistake the malaria of older children for tubercular meningitis. In the first two years of life, however, malaria may affect so insidiously the general nutrition before its characteristic symptoms appear that some doubt as to the differential diagnosis may arise. The following case illustrates this fact :

A male infant (Case 268), twenty months old, with a history of tuberculosis on the mother's side, began to show symptoms of anæmia and malnutrition with no perceptible cause, such as either improper food or bad general hygiene, to account for it. After two or three weeks it had attacks of unconsciousness lasting for hours; at other times drowsiness, with irregular pulse and respirations, was present. The temperature was  $39.5^{\circ}$  to  $40^{\circ}$  C. ( $103^{\circ}$  to  $104^{\circ}$  F.). There were slight convulsions, and the fontanelles were distended. At first there was no periodicity of the symptoms, but a week later the attacks were evidently more pronounced every other day, and the infant was brighter on the intervening days. It lived in a malarial district.

On the administration of quinine and on removing the infant to a non-malarial region, these symptoms entirely disappeared.

The detection of the plasmodium would, of course, have determined the diagnosis in this case, but it could not be obtained.

Another case, which I saw in consultation with Dr. Parker, of Princeton, is also very instructive in warning us how careful we should be in making a diagnosis of tubercular meningitis in cases where there is a possibility of malaria being the cause of the symptoms.

A male infant (Case 269), fourteen months old, had always been well until fourteen days previous to the time when I first saw it. It then began to be fretful and to have diarrhœa. This condition continued for about a week, when it fell into a stupor, became very anæmic, and it was necessary to feed it by means of a dropper. At times it would cry out sharply. The temperature varied from  $37.2^{\circ}$  to  $38.7^{\circ}$  C. ( $99^{\circ}$  to  $102^{\circ}$  F.). The respirations were usually regular, but at times were of the Cheyne-Stokes type. The pulse was about 120, sometimes regular, but at times intermitting. The pupils were sometimes contracted, but showed no irregularity. No other abnormal conditions were noticed, such as paralysis or symptoms connected with the lung, ear, heart, or throat, but the abdomen during the twenty-four hours previous to my examination was beginning to be depressed. The *tache cérébrale* was very distinct.

On close inquiry I found that there was a slight periodicity in the symptoms, shown by a rise of temperature on each afternoon and followed by the stupor becoming somewhat less. Although the infant had been unconscious for a week, and was becoming weaker and taking less nourishment every day, yet, on the supposition that it might possibly be an obscure case of malaria, I decided that quinine should be administered in suppositories. On the next day a slight improvement was noticed in the infant in the afternoon. It appeared less comatose, but its temperature and pulse remained as on the previous days. On the following day, which was the second from the time that it had begun to receive the quinine, it rapidly became conscious and began to drink milk. On the following days it was reported to have had a restless night and to have had two slight convulsions. Its temperature in the morning was  $38.2^{\circ}$  C. ( $100.9^{\circ}$  F.), and the pulse was 115 and not intermittent. On the following day there was marked improvement in every way, and this continued without interruption for the next four days. The infant then continued to improve rapidly, the temperature and pulse becoming normal, and some months later it was reported to be perfectly well.

(5.) **Typhoid Fever.**—In my experience typhoid fever in young children is the disease which, next to non-tubercular meningitis, is most likely to simulate and be mistaken for tubercular meningitis. We may also have considerable difficulty in differentiating tubercular meningitis from the non-tubercular meningitis which may occur in the course of typhoid fever. The extreme cerebral congestion which at times arises as a symptom of typhoid fever may also add fresh difficulties to the differential diagnosis. The



decisive point, however, between typhoid fever and meningitis, whether tubercular or non-tubercular, is the absence of leucocytosis in typhoid fever and its presence in meningitis, provided that the latter is to any degree purulent.

According to E. S. Wood, in meningitis the chlorides in the urine diminish rapidly; heating the urine precipitates the phosphates readily, and the amount of indoxyl is increased: the reverse of these reactions occurs in typhoid fever.

(6.) **Syphilis.**—The history and general symptoms of syphilis are to be sought for where a syphilitic meningitis is suspected. The temperature is not especially high, and the symptoms are seldom acute. The pathology is said to be usually that of a chronic basic meningitis.

(7.) **Rheumatism.**—Rheumatism is said to occur as a cause of meningitis, but this must be rare, and I shall merely mention it, as I have never met with a case of this kind. A high temperature and acute symptoms are said to be the rule in rheumatic meningitis.

(8.) **Nephritis.**—In addition to the other diseases which may simulate tubercular meningitis should be mentioned nephritis, in which the symptoms of uræmia simulate, to a certain extent, those of tubercular meningitis. The urine should always be examined in doubtful cases of this kind, as where uræmic symptoms resulting from nephritis are present the disease will be shown by such examination, and we shall thus be able to differentiate it from tubercular meningitis.

(9.) **Cerebro-Spinal Meningitis.**—It is often quite difficult to differentiate the early stages of tubercular meningitis from those of cerebro-spinal meningitis. In typical cases, however, the diagnosis is easily made, as the long prodromal period of tubercular meningitis, as a rule, does not occur in cerebro-spinal meningitis, and the temperature in the latter disease is almost always high, while in the former it is, as I have already told you, raised to only a moderate degree. In fact, all the symptoms of cerebro-spinal meningitis are markedly acute in comparison with those of tubercular meningitis, which is essentially a disease of a subacute character. I shall presently show you a case of tubercular meningitis (Case 272, page 618) which simulated cerebro-spinal meningitis very closely.

(10.) **Non-Tubercular Meningitis.**—On closely studying what I have already told you of the symptoms of meningitis in general, you will be able in the great majority of cases to differentiate it from other diseases, provided that you do not attempt to make the diagnosis too early. Remember that you are seldom warranted in making an early diagnosis, in view of the wide range of possible nervous symptoms which can be met with in young children. Having determined that the disease is of cerebral origin, we must next differentiate between the tubercular and non-tubercular forms of meningitis by means of the broad rules of which I have just spoken, and which I have condensed and simplified by means of this table (Table 101, page 612).



TABLE 101.

CEREBRAL MENINGITIS.	
Non-tubercular.	Tubercular.
Usually secondary (possibly primary).	Secondary.
Not hereditary.	Hereditary.
Acute.	Subacute.
Prodromata short, if any.	Prodromata long, decided.
Headache severe at once, with delirium early, and soon followed by somnolence.	Headache less severe at first, but gradually increasing; delirium less common and milder.
Photophobia extreme.	Photophobia not so marked.
Convulsions violent.	Convulsions less violent.
Temperature high.	Temperature moderate.
Pulse and respiration rapid.	Pulse and respiration slow and irregular.
Duration short.	Duration long.

Transudation into the ventricles may occur in either form. The younger the infant the nearer the two forms approach each other in the similarity of their symptoms. Caillé has lately shown the value for diagnosis of Quincke's method of tapping the spinal canal.

**INFANTILE TUBERCULAR MENINGITIS.**—According to some extended observations made at the hospital in Stockholm, infantile tubercular meningitis is characterized in the first year by an absence of prodromata, the sudden development of acute symptoms, a short course, and a fatal issue. The temperature is high,  $38.8^{\circ}$ – $39.4^{\circ}$ – $40^{\circ}$  C. ( $102^{\circ}$ – $103^{\circ}$ – $104^{\circ}$  F.). The respirations are quickened and comparatively regular, 30–40–50. The pulse is high, 130–140–150. Clonic spasms and strabismus often occur. Paralysis is quite frequent, and diarrhoea is present rather than constipation. Bulging of the fontanelles is usual. Sinking of the abdomen is rare. Vomiting may occur, but is not especially common. Sharp cries are occasionally met with. The differential diagnosis from non-tubercular meningitis is difficult. Sopor and coma at the end are frequent in both diseases. The duration is seldom more than a week. It may be only two days, yet in rare cases the infant, like the child, may live for a month.

During the second year the symptoms of tubercular meningitis become of rather an irregular type, sometimes assuming the character of those which are seen in the first year, but soon corresponding more nearly to those which are met with in the middle period of childhood.

**PROGNOSIS.**—Where we are sure of our diagnosis, I believe that in our prognosis we should give no hope of recovery whatever, except that in the extremely rare cases which I have just mentioned a temporary remission may take place. The reported cases of absolute recovery from tubercular meningitis cannot but be looked upon with scepticism. Indeed, the non-tubercular forms of meningitis simulate the tubercular so closely that without post-mortem verification recoveries can be supposed to be possible, but can hardly be accepted as proved.

**TREATMENT.**—The treatment of tubercular meningitis up to the time when the diagnosis is established should be purely symptomatic; later we

should make the child comfortable by every means in our power. As no case of tubercular meningitis has ever been proved to be cured by iodide of potassium or any other drug, it is useless and unwise to encourage ourselves and the parents by false hopes of good results arising from the administration of any drug whatever. Up to the present time our knowledge of the disease justifies us only in using drugs as palliatives for the child's suffering.

The following case illustrates very well the tubercular meningitis of middle childhood:

A boy (Case 270), five years old, had always been well and strong. On December 3, while endeavoring to climb into bed, he fell and struck the back of his head. He cried afterwards, but the blow left no mark, and nothing was thought of it. The following day, while playing, he fell and struck the back of his head, but the blow was no more serious than he had often had before. On the next evening he went to a children's party, ate nothing unusual, went to bed early, and slept all night. On the following day he was unable to eat and was somewhat fretful, both of which conditions were unusual for him.

On December 29 he had a slight follicular tonsillitis. His pulse and temperature were normal, the cheeks were flushed, the eyes dull, and the pupils normal. His head was slightly hot, and he was dull and drowsy. He did not have any movement of the bowels for two days, but on the third day they were moved by means of medicine. He continued to be in about the same condition until January 2, when his temperature was  $37.2^{\circ}$  C. ( $99^{\circ}$  F.), and his pulse 64, regular and strong; his face was flushed, and his eyes were vacant and staring. He vomited once on that night, passed his water involuntarily, moved his left leg spasmodically, and clinched his hands occasionally. He was evidently uneasy, and moaned a good deal.

On the following day the pulse was occasionally intermittent. In the mean time he became more and more drowsy, and finally relapsed into a state of unconsciousness.

On the 3d of January the pupils were normal, but he was completely unconscious. The temperature was  $38.1^{\circ}$  C. ( $100.6^{\circ}$  F.), the pulse 180, and the respirations 30.

I saw the child on January 4, and on making a careful physical examination found nothing abnormal, except a slight congestion of the ear in the neighborhood of the malleus, and in the back over the apex of the lung was a slight elevation of pitch on percussion. The temperature was  $39.1^{\circ}$  C. ( $101.2^{\circ}$  F.), and the pulse was 89 and strong. There was considerable twitching of the arms, chiefly on the right side, lasting from ten to twenty minutes. The pupils were slightly contracted, but were alike. That night he drew his right hand across the face with a quick trembling motion, the right leg being drawn up and the whole body trembling; occasionally there was moaning and sighing respiration.

Dr. C. J. Blake, who examined the ears, reported that there was a slight congestion in the posterior canal of both ears and also in the neighborhood of the right malleus. Both membranæ tympani were clear, normal, transparent, and without injection of the manubrial blood-vessels. There was, in fact, no evidence of disturbance of the ears. On the posterior wall of each external auditory canal at the anterior third, more pronounced in the right than in the left ear, was a circumscribed patch of injection such as is observed in cases of inflammatory process in the mastoid antrum, and occasionally uncomplicated congestion of the middle ear. Dr. Blake thought that the congestion was merely a symptom of the meningeal congestion and was not the cause of the disease.

During the next few days the boy's condition varied but little. The eyes, usually closed, would at times open completely, when the eyeballs could be seen to move from side to side. The respiration was sighing, interrupted, occasionally almost inaudible, and then for a time noisy. At times the breathing was suspended for several minutes, when bright red spots would appear on the cheeks; these would disappear when the respiration was resumed. The patient moaned occasionally, and there was some twitching and moving of the extremities, but no convulsions. The pulse was fair in strength, but at times intermittent. The temperature varied, but was moderate in degree.



The extremities of the right side were absolutely motionless, and sensation was apparently absent. The child lay, as a rule, perfectly quiet, as though asleep, and at times would present the picture of a perfectly healthy child sleeping.

On January 9 the extremities became cold, the face very pale, and the pulse imperceptible. This condition lasted fifteen minutes, when he improved in appearance. During the night the breathing grew very rapid, he was restless, moved the left arm continually, and moaned. After some time he opened his eyes, looked around the room, and then became quiet and slept. The next day he was slightly unconscious, and the fingers were flexed, with a very strong contraction of the muscles. The breathing then became more difficult, the nostrils being widely dilated with every breath. During the night he was conscious for some time, swallowed water without difficulty, and the eyes were wide open.

On January 11 there was ptosis of the right eyelid. The pulse became regular, compressible, and intermittent. The left arm was occasionally raised to the head with a quick spasmodic motion, the child moaning as if distressed. Later the eyes became fixed, the pupils dilated, the nostrils expanded, and a bluish color appeared around his mouth and nose. The breathing became very difficult. During an attack of this kind he had every appearance of being moribund, and each attack was thought to be his last.

The change from day to day in the child's general condition was almost imperceptible. He was, however, gradually becoming emaciated.

On January 12 the pupils of both eyes were much dilated; the right eye was almost motionless, with ptosis of the right lid, while the left eye moved occasionally from side to side in a circle. The face was livid, and the hands were mottled with bright red spots. Later, the left eye became quiet and had a slightly contracted pupil.

On the following day, January 13, the movements of the left eye were repeated, the right pupil being dilated, while the left one was contracted. During all this time the enemata were retained, the bowels moved regularly, and the urine was passed normally. The pulse was so weak that at times it could not be found at all at the wrist, and the breathing was at times inaudible and almost imperceptible.

On January 17 there was slight discharge of pus from the mouth, and also from the left eye. During the next day his breathing grew more and more difficult, and it seemed as though he could not possibly live much longer. In the evening, however, his respiration was much easier and his whole appearance was greatly improved. His breath was very offensive, and there was a loud bubbling sound in the throat.

On January 19 the right nostril was much more dilated during inspiration than the left. The forehead was shiny and slightly œdematous, and the veins were plainly mapped out. Occasionally he moved his right hip-joint and shoulder, which had been motionless for days. There was another slight discharge of pus from the mouth, and when his lips were wiped he seemed more sensitive to touch than before. During the night his left arm and left leg were constantly moved, and he moaned as though he were still in pain. His forehead was still œdematous.

During the next day he was in a state of deep coma for four hours. He then drew a deep sigh and seemed somewhat conscious. The pulse was soft, intermittent, and fluctuating.

On January 20 he partly opened and shut his right eye, which was very sensitive to light. The breathing was difficult and noisy. The face was covered with perspiration. At 10 P.M. the sighing respiration began again, and at 10.15 he died quietly, on the thirty-first day of the disease.

The autopsy was made eighteen hours after death by Dr. W. W. Gannett, and the report was as follows:

The body was much emaciated. There was slight lividity of the dependent portions. Rigor mortis was present. Nothing unusual was observed about the calvaria or dura mater. The sinuses of the latter contained partly congested blood. The pia mater of the convexities of the brain was very dry, and the minute vessels were injected. The convolutions were flattened. The sulci were obliterated. The pia of the base along the chiasma and in the fissures of Sylvius was thickened, and there was an opaque yellowish-gray color from the presence of a fibrino-purulent material in its meshes. In the above situations,



also on the under surface of the frontal and temporal lobes, also on the pons and inner borders of the occipital lobes, were to be seen very numerous, gray, translucent nodules about 2 mm. ( $\frac{1}{8}$  inch) in diameter. The lateral ventricles each contained about 50 c.c. ( $1\frac{2}{3}$  ounces) of a slightly opaque fluid. The ependyma was thick, grayish, and opaque. The choroid plexuses and velum interpositum were markedly injected. In the latter were to be seen several small nodules similar to those described in connection with the pia of the base.

A section of the hemispheres showed nothing remarkable, the puncta cruenta being of about the usual size and number.

The basal ganglia, pons, medulla, and cerebellum also showed no appearances worthy of special note.

The heart was normal.

The pleural surfaces on both sides were free from adhesions; the pleural cavities contained no fluid.

Both lungs retracted readily, and were crepitant everywhere except at the apices, where small nodules could be felt within the tissue.

On section an opaque, grayish-yellow, cheesy nodule, 6 mm. ( $\frac{1}{4}$  inch) in diameter, surrounded by a narrow border of gray and translucent tissue, was found at the top of the left lung. At the top of the right lung were several closely aggregated nodules of a similar appearance, forming together a mass about 2.5 cm. (1 inch) in diameter. The other portions of the lungs were normal.

The bronchial lymph-glands were enlarged to 1.2 cm. ( $\frac{1}{2}$  inch), showing on section a yellow, opaque, crumbling material.

The spleen was of the usual size, color, and density. On section the follicles and trabeculae were found to be fairly distinct; the pulp was firm and of a pale red color. Two or three gray, translucent, sharply defined, slightly projecting nodules, 1 mm. ( $\frac{1}{25}$  inch) in diameter, were to be seen. The kidneys were normal. In the lower third of the ileum a loss of substance of the mucous membrane was found in several places. The edges of these lesions were elevated and their bases granular. The liver was found to be normal.

The **pathological diagnosis** was—

- Tubercular meningitis,
- Acute hydrocephalus,
- Ependymitis,
- Tuberculosis of the velum interpositum,
- Tubercular nodules in the lungs,
- Tuberculosis of the bronchial lymph-glands,
- Tuberculosis of the spleen,
- Tubercular ulcerations of the intestines.

I have here in Bed 3 an interesting case of tubercular meningitis to show you.

This boy (Case 271) is three years old. There is no history of tubercular or syphilitic disease in the parents.

Three weeks before entering the hospital, the child, who had previously been healthy, began to complain of pain in the abdomen, and to have anorexia and a feeling of general malaise. Somewhat later it was noticed that the eyes would at times turn inwards and that the head would be drawn back. He was in this condition for two weeks before entering the hospital.

On March 13 he was brought to the hospital, and was found to have a temperature of 38.4° C. (101.2° F.), a pulse of 120 and not intermitting; the respirations were 40. He was in an unconscious and drowsy condition. His head was drawn back, and he did not wish to lie on his back. The tongue was not coated. An examination of the heart, lungs, and urine showed nothing abnormal. An examination of the eyes, made by Professor O. F. Wadsworth, showed the pupils to be dilated, but equal in size and reacting to light. There was internal strabismus of both eyes. There was optic neuritis and the beginning of

an atrophy following the neuritis. The patellar reflexes were absent, and there was no ankle-clonus. The superficial reflexes were normal. There was no tenderness of the head or spine. An examination of the ear, which was made by Professor J. O. Green, showed nothing abnormal.

On March 16, as you remember, I examined the child before you with Dr. Bullard. At this time he showed nystagmus with conjugate deviation to the right or to the left, according to the side on which he lay. No *tache cérébrale* was found.

On the 17th an erythematous congestion was noticed on the right cheek, and he became still more somnolent.

## CASE 269.



Tubercular meningitis. Male, 3 years old.

On the 18th the head was much less retracted. He had vomited once during the night and once in the morning.

On the 21st he had a convulsion, which was the first that had occurred during the course of the illness. He was also found to have partial opisthotonos. The legs did not participate in the contraction, but the head was drawn back almost to the buttocks. He was found to have Cheyne-Stokes respiration. During this day he had four or five convulsive attacks, and remained in a condition of opisthotonos in the intervals between the attacks. These convulsive attacks lasted about half a minute each, and the intervals between them were about four minutes. There was incontinence of urine and of feces. The pulse was rapid and irregular, and the extremities were cold. The *tache cérébrale* was obtained on this day, and lasted for twelve minutes. 0.12 gramme (2 grains) of chloral and 2 grammes ( $\frac{1}{2}$  drachm) of brandy were given subcutaneously. The convulsions ceased, the opisthotonos disappeared in twenty minutes, and the child remained quiet.

On the 26th the record was that for two days the child had been decidedly better, the retraction and strabismus were less, the nystagmus had disappeared, and he had recognized and spoken to his father. The *tache cérébrale* could be obtained, but was less distinct, and the temperature was normal.

On the 28th he became worse again. His head was again retracted, but he was not wholly unconscious. There was retention of urine, for which he had to be catheterized.

On the 29th he had a convulsion lasting three minutes, in which the right arm was jerked up over his head. This was followed by partial opisthotonos, and then by a general convulsion lasting two or three minutes, during which his eyes rolled up. At times he would have convulsive movements and tremor without actual convulsions.

To-day (April 7), as you will notice, the right hand lies motionless by his side and is in a state of extreme pronation. He is unconscious, and all the abnormal symptoms have



returned. You see that he has the characteristic aspect of a typical case of tubercular meningitis. The eyes are open and staring, the head is drawn back, the abdomen is retracted, and on drawing the finger over the thigh you see the *tache cérébrale* is very marked. The respirations are of the Cheyne-Stokes type, the pulse is intermittent. The temperature has varied from 37.2° to 38.8° C. (99° to 102° F.), but has risen within the last twelve hours to 40° C. (104° F.), which indicates that the fatal issue of the case is very near.

(Subsequent history of the case.) On the following day there were a number of convulsions occurring in rapid succession, especially involving the left side. The child groaned and sighed a number of times; his arms and legs were rigid, his eyes were rolled upwards. At two o'clock in the morning he took some milk, but after that refused it, and from that time until his death, at 7.25 A.M., he was in a condition of continued convulsions.

I shall now ask you to come to the autopsy-room, in order that you may see the results of the post-mortem examinations of some children who have died of tubercular meningitis. Dr. Gannett has some specimens here to show you of a case which has just died in the hospital. When the patient was alive the case simulated cerebro-spinal meningitis very closely, and you have already seen it in the wards. It is a very instructive case, as it is an unusual one, and illustrates an important fact in connection with tubercular meningitis,—namely, that the patient may recover temporarily from an attack of the disease and finally die of a recurrent attack. This is, however, a very rare occurrence.

You may remember that when this infant (Case 272) was alive I explained to you the difficulties which may arise in making a definite diagnosis in cases where cerebral symptoms are present.

It was twenty-one months old when it entered the hospital. The history obtained from the mother was that she had always been healthy, but that the father was supposed to have had the primary lesion of syphilis three years previously, although no secondary manifestations had appeared. The infant was born after an unusually long labor with a prolonged forceps delivery.

It was stated to have been healthy until it was nine months old. At that time it had a convulsion, which first affected the right and then the left side. It was unconscious for ten days, and was somnolent for four weeks. Two or three weeks later its general condition improved. During this time the infant did not use the muscles of its left side or limbs, and it could laugh only with the right side of its face. Its body was turned continuously to the left; sensation was not interfered with. It gained slowly in strength, and the symptoms gradually disappeared, until it was thirteen months old, when it seemed to be comparatively well, all motor disturbances having ceased. In the following months it had a few slight attacks of the same nature. The final attack from which it died occurred when it was twenty months old, and began with a convulsion on the right side with twitching of the muscles on the left side and frothing at the mouth. There was also ptosis of the left eye. It did not cry out when going into the convulsions, but had marked opisthotonos, which lasted, to a greater or less extent, for five weeks. During these five weeks it was unconscious, and there were several slighter attacks.

On entering the hospital, physical examination showed that the infant was of medium size, pale, poorly developed and nourished, and unable to stand, the left leg being weaker than the right. Nothing abnormal was found in connection with the heart or lungs. She could use her extremities partially, but there was an evident motor disturbance of the whole of the left side, and she took hold of objects with her right hand only. The index and little finger of the left hand were frequently found to be extended, the second and third fingers being flexed partially. There was also slight drooping of the left eyelid, and the lines of the left side of the face were obliterated. There was a very slight drooping of



the left corner of the mouth. There was slight strabismus of the left eye, and an apparent lack of power of the left external rectus muscle. The patellar reflexes were exaggerated on the left side. Examination of the ankle-clonus was negative. The epiphyses of the wrists were somewhat enlarged. The child could not speak, and apparently could not understand readily. No evidence of a history of cerebral injury could be obtained. The circumference of the chest was 1 em. ( $\frac{3}{8}$  inch) larger than that of the head. The cause of the disease was so obscure that at this period the diagnosis could not be definitely made, the supposition being that the child was suffering from the results of an attack of cerebro-spinal meningitis, or possibly from tertiary syphilis, or that a cerebral hemorrhage had taken place, with a resulting spastic paralysis.

While in the hospital the child presented a number of different nervous phenomena. At times she would appear to be for days semi-comatose and would not take notice of anything about her; the eyes rolled up and she would have slight twitching of the body, but this was not localized, and there were no convulsions. At another time, while sleeping quietly during the night, she was found to be unconscious in the morning, and to have her head slightly drawn back and her eyes turned up. Nystagmus was present, and the pupils were dilated and did not react to light, but were equal in size. Clonic twitching of the right foot and the muscles of the right side, flexion of the fingers of the right hand over

## CASE 272.



Recurrent tubercular meningitis. Female, 21 months old.

the thumb, and twitching of the muscles of the wrist sometimes occurred. There was twitching of the fibres of the sterno-mastoid muscle on the right side. There was also twitching of the right side of the face. There was no spasm on the left side, except of the left sterno-mastoid, but there was a nystagmus of the left eye. These clonic twitchings were rhythmical and occurred 180 times a minute. The pulse was 172, and was very feeble. The respirations were 80, rapid and rattling; the temperature was 39.4° C. (103° F.).

From 2 A.M. until 5 A.M. 0.36 gramme (6 grains) of chloral was given by enema, and 0.36 gramme (6 grains) of bromide of potassium was given every three-quarters of an hour by the mouth, alternating with the chloral. The spasms became less marked after 3 A.M., but continued in a mild degree up to 11 A.M. During the remainder of the day the child lay in a stupor, but had no convulsions. It was able to swallow brandy and milk, which were given to it by the mouth in small quantities at different intervals.

On the day following this attack the report was that the child had slept well, and that there was more or less stupor, but there had been no convulsions.

On the following day the condition remained about the same, but on the next day she apparently had attacks of pain, when she would straighten herself out, throw back her head, and cry out.

On the following day, about 11 A.M., she began to have the same twitchings as in the attack previously mentioned. They were of the same character, except that the extensor

muscles of the left foot contracted feebly. The convulsions ceased at about 2 P.M., and the child remained in a stupor.

On the following day it was reported that she had had no convulsions, but apparent attacks of pain, when she would cry out and throw her head back, and that she had had an attack of opisthotonos, in which condition you will remember you once saw her. This condition of opisthotonos at times would be much more marked than when you saw her, so that the heels would almost touch the back of her head. The next symptom which appeared was stupor. The temperature at this time was considerably elevated.

On the following day there were no convulsions, and her condition was about the same as on the previous day, but the head was drawn back and was rigid, and the legs were drawn up and were held rigidly. She lay in this condition, most of the time in a stupor, crying out occasionally, and moving her left hand and arm more than she did the right. At times she would appear to be sleeping naturally and the rigidity would pass away.

The opisthotonos gradually became more marked and more frequent in its occurrence, and, although the bowels were moved regularly every day, she took less nourishment, and the temperature continued to rise, and varied from  $37.7^{\circ}$  to  $40^{\circ}$  C. ( $100^{\circ}$  to  $104^{\circ}$  F.).

The time when you saw her in the condition of opisthotonos was the sixth week from the time of this last attack. During the last week of her life the opisthotonos became less marked, and at times passed away entirely. She opened her eyes, but the pupils reacted very slightly. The left pupil became somewhat larger than the right and reacted slightly, while the right pupil did not react at all. The spastic condition of the right wrist and left knee persisted, the patellar reflexes were equal and normal, and the child lay in a semi-stupor, with a temperature varying from  $38.3^{\circ}$  to  $39.4^{\circ}$  C. ( $101^{\circ}$  to  $103^{\circ}$  F.). She took less and less nourishment, and had a slight cough. She gradually lost in weight and became weaker, and on the day before she died her respirations for a time were very rapid, running up to 100 a minute. Death took place apparently from exhaustion.

The long duration of this last attack, embracing a period of eight or nine weeks, made the diagnosis very difficult, and prevented us from making the clinical diagnosis of tubercular meningitis, which these specimens just found at the autopsy prove to be the disease by which the child was affected from the beginning.

This chart (Chart 21, page 620) represents the temperature, pulse, and respirations of this case during the last twenty-one days of its life.

On examining the brain you see that the dura mater is normal, the pia mater of the convexities is pale, and the cerebral convolutions are somewhat flattened. The pia mater at the base of the brain shows considerable infiltration with fibrin, which is quite firm, but there is little or no injection of the blood-vessels. In many places in the portions of the pia mater at the base of the brain where the meshes of the pia are not infiltrated with fibrin, gray nodules as large as a pin-head are to be seen. The lateral ventricles are at least six times the usual size, the layer of brain-substance between the cavity and the convexity being considerably thickened. The ependyma of the lateral and fourth ventricles is thickened and granular. On section the brain-substance is found to be pale, and the puncta cruenta small. Sections of the basal ganglia, pons, medulla, and cerebellum show that the brain-substance is normal. The spinal cord shows in gross nothing unnatural. The heart is normal. Beneath the pleura of both lungs numerous gray nodules the size of pin-heads are to be seen. At the apex of the left lung is a cheesy nodule 0.5 cm. ( $\frac{1}{2}$  inch) in diameter. Both lungs are extensively studded with gray miliary tubercles. The spleen and kidneys show similar appearances, and the bronchial and lymph glands are very much enlarged and show throughout their substance cheesy degeneration.

The **pathological diagnosis** in this case is—

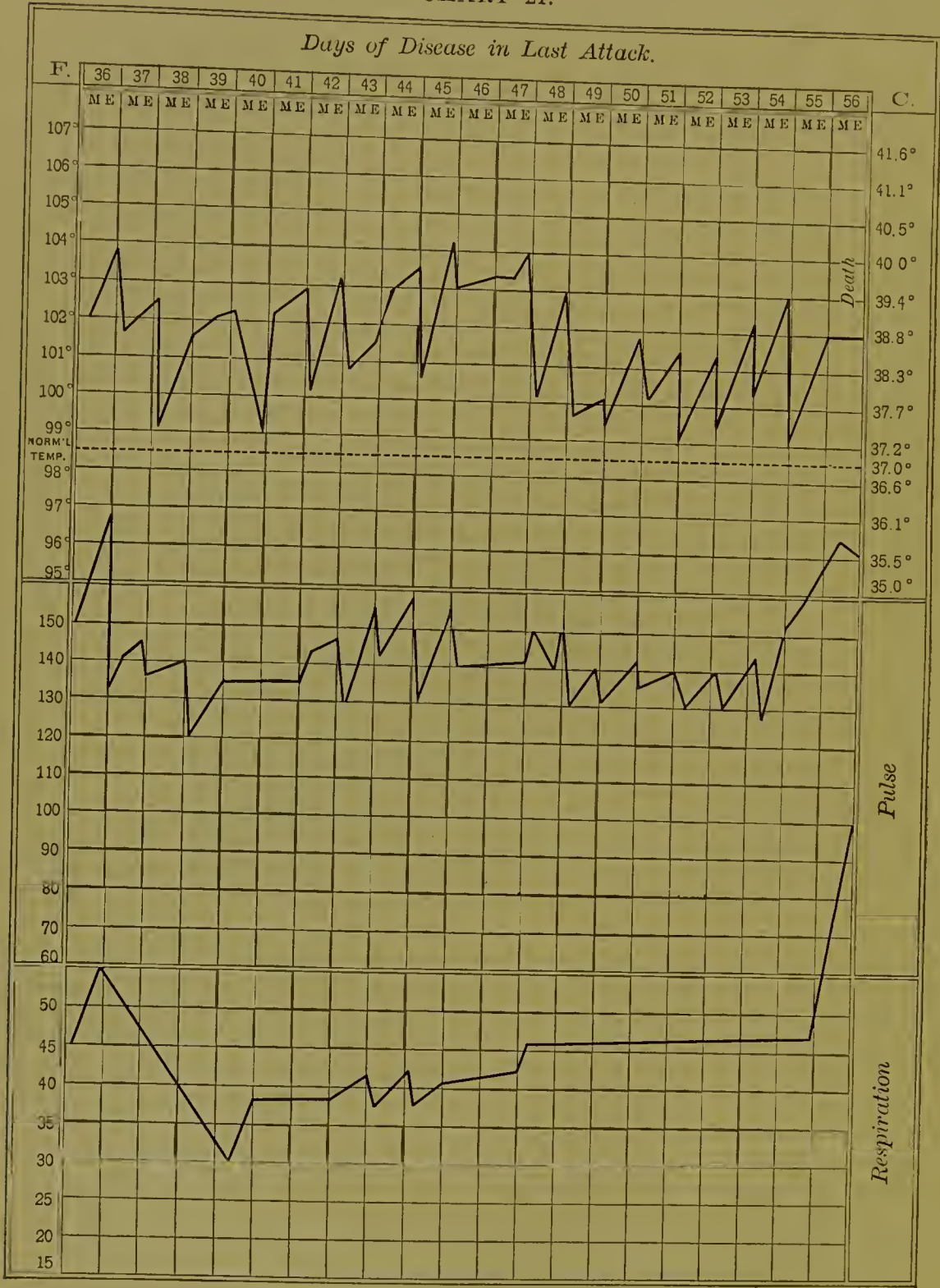
- Subacute tubercular meningitis,
- Chronic granular ependymitis,
- Chronic hydrocephalus,
- Atrophy of the brain-substance,
- Miliary tuberculosis of the lungs, spleen, and kidneys,
- Chronic tuberculosis of the lung.

As Dr. Gannett has explained to you, some of the tubercular lesions are of recent



growth, while others are evidently old ones and representative of a former attack. You see, therefore, that the presence of older tubercular lesions in the meninges, as well as of

CHART 21.



Recurrent tubercular meningitis. Last 21 days of life.

those which produced the symptoms in the last attack from which the infant died, proves to us that the case is one of recurrent tubercular meningitis.

These cases of recurrent tubercular meningitis are rather rare, and the disease is so uniformly fatal in the first attack that I shall recall to your



minds the case which was under Dr. Townsend's care at the Good Samaritan Hospital.

A little girl (Case 273), five years old, entered the Good Samaritan Hospital with hip-disease on the left side and dorsal Pott's disease. She was treated in bed for these diseases, and did very well for a time, but on May 7, after a week in which she showed anorexia and loss of weight, she began to vomit, and on the following day she complained of headache and photophobia. She rolled her head from side to side. Her bowels were constipated, and could not be moved by enemata, and her abdomen was much retracted. This continued for four days, with at times delirium, accompanied by marked drowsiness. There were also ptosis of the left eyelid, slight convulsive movements of the limbs, and frequent putting of her hands to her head, as though she were in pain.

On May 12 she had recovered so much that she played with the other children and called for her books and toys. The left pupil, however, remained a little smaller than the right.

On the 15th of May, and again on the 20th, 21st, 25th, and 27th, the patient became drowsy, and complained of headache. In the intervals between these attacks she seemed bright and well. During the drowsy periods her abdomen was retracted and her bowels were constipated.

From the 27th of May until the 20th of July she appeared as well as usual. On the latter date her temperature suddenly rose to  $40.1^{\circ}$  C. ( $104.2^{\circ}$  F.). She had pain in the head and photophobia, and the right pupil was larger than the left. This lasted only two days. She then became bright and well again, and continued so for over ten weeks.

On October 2, having been perfectly well on the previous day, she began to vomit and to complain of headache. Two days later she fell into a stupor and became completely comatose.

On October 6 the left pupil was widely dilated and the right one was contracted to the size of 2 mm. ( $\frac{1}{12}$  inch); there were convulsive movements, and later in the day she died.

The post-mortem examination showed a recent tubercular meningitis. In addition to these lesions there were found some older large tubercles of the brain and the remains of the previous attacks of tubercular meningitis.

Nothing else of importance was detected in the other organs.

I will now ask you to return to the wards, to see a case of tubercular meningitis in a child, two and a half years old, who entered the hospital at what was supposed to be about the tenth day of the disease.

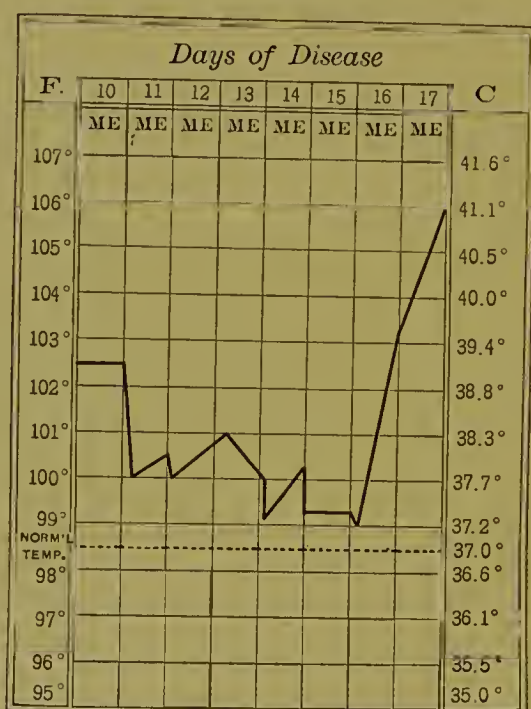
The history of the case (Case 274) is that the father's mother and the mother's mother and brother died of consumption. When this child was one year old he had measles, otherwise he had always been well. About two or three weeks ago it was noticed that the child slept more than usual. At that time he appeared to be feverish and his tongue was noticed to be coated, but there was no nausea nor vomiting. A few days later he vomited once or twice during the day. The bowels were constipated. Eight days before entering the hospital he had a slight convulsion, and three days later he cried a great deal, as if he were in pain. Two days before entering the hospital he had a number of convulsions during the night, each lasting about ten minutes. On the following day the convulsions occurred again. On the day he entered the hospital he began to have convulsions at three o'clock, which lasted about two and a half hours. He was also noticed to have marked internal strabismus of the left eye and slight strabismus of the right eye. The muscles of the neck were somewhat contracted. There was no paralysis of the extremities.

The pupils were equal, they reacted to light, and were somewhat dilated. The conjunctivæ were injected, the left one especially so. Sensation was not impaired. The knee-jerks and ankle-clonus were absent. There was a marked *tache cérébrale*. The respirations were irregular and sometimes of the Cheyne-Stokes type. The child was unconscious and was very pale. The heart was found to be beating very rapidly, sometimes as high as 200

beats in a minute. No souffles were detected. The temperature was  $38.3^{\circ}\text{C}$ . ( $101^{\circ}\text{F}$ .). During the next day the child lay in a state of stupor. He continually moved the left forefinger and thumb, kept drawing the head to the left, and was very restless. He was reported to have cried all night and to have put his hand to his left ear. He lay with his eyes wide open, took nourishment well, and had less strabismus than when he entered the hospital. On the following day (about the thirteenth day of the disease) he was very restless, had sordes on the teeth, and his tongue was very dry. Examination of the ears showed nothing abnormal. The abdomen was somewhat retracted. He was less restless, and slept a good deal. The bowels were moved regularly, and the movements appeared to be well digested. He took about 90 c.e. (3 ounces) of milk every two hours. On the following day there was no especial change, except that the muscles of the neck were firmly contracted and the *tache cérébrale* came out more slowly than on the previous day. A slight paralysis of the left side of the face appeared on this day. The left eyelid moved rather slowly, and the left corner of the mouth seemed to drop a little. The pulse was irregular, of fair strength, and intermittent. He did not take his nourishment so well. Yesterday the child was in about the same condition.

To-day you see that he is lying in a comatose condition, with his eyes half closed. The pupils are rather irregular, dilated, and do not respond to light. The face is somewhat cyanotic, especially about the nose and the eyes. The respiration is decidedly of the Cheyne-Stokes type. The pulse is irregular and intermittent. On drawing my finger over his thigh you see that the *tache cérébrale* is well marked and that it lasts about ten or fifteen minutes. The head is somewhat drawn back. What I wish especially to call your attention to is the temperature chart (Chart 22). You will notice that the tempera-

CHART 22.



Tubercular meningitis. Male,  $2\frac{1}{2}$  years old.

ture had risen yesterday from  $37.5^{\circ}\text{C}$ . ( $99.5^{\circ}\text{F}$ .) to  $39.5^{\circ}\text{C}$ . ( $103.2^{\circ}\text{F}$ .), and that it is now rapidly rising until it has reached  $41.1^{\circ}\text{C}$ . ( $106^{\circ}\text{F}$ .).

This rise of temperature is very significant, and denotes that the child will die very soon.

(Subsequent history.) The child died quietly on the evening of what was supposed to be the seventeenth day of the disease.

The autopsy was made by Dr. Mallory. Rigor mortis was present; the left pupil was dilated; there was moderate lividity of the dependent portions of the body.

**Heart.**—The right ventricle was dilated and contained dark, clotted blood. The valves were normal.



**Lungs.**—A number of small, flattened, gray masses were found in the pleura; on section they were found to be miliary tubercles. The right lung was adherent to the parietal pleura by strong fibrinous adhesions, beneath which were miliary tubercles, especially in the areas covering the ribs, the diaphragm, and the upper third of the sternum. A small number were also found in the substance of the lung. The bronchial glands were enlarged, one of them being 1.2 cm. ( $\frac{1}{2}$  inch) in diameter. This gland on section was yellow and somewhat broken down.

**Spleen.**—The spleen was of about normal size and showed many flattened miliary tubercles. Beneath the capsule, on section, there were found numerous tubercles of varying size: the larger ones were yellow and the smaller ones gray.

**Peritoneum.**—There were found scattered all through the omentum, on the surface of the root of the mesentery, over the bladder, and particularly on the under surface of the right side of the diaphragm, numerous miliary tubercles. The lymph-glands of the mesentery were considerably enlarged, particularly beneath the stomach. On section they showed tubercles, most of which were quite large and had yellow, cheesy centres.

**Intestine.**—In the intestine about the ileo-cæcal valve there were several small ulcerations apparently in the process of repair. In the cæcum there were two narrow ulcers about 1.5 cm. ( $\frac{5}{8}$  inch) long. The bases were injected. The walls were not broken down.

**Liver.**—Many rather large tubercles were found beneath the capsule of the liver. They were flat, but not cheesy.

**Brain.**—The convolutions of the brain were flattened. There was marked fibrino-serous exudation at the base of the brain, covering the optic commissures and the adjoining parts. The third nerve was chiefly injected. Many small tubercles were present in the fissures of Sylvius and over the convexities of the brain. In the right half of the cerebellum, just beneath the pia, about the centre of the base, was a yellow nodule about 6 mm. ( $\frac{1}{4}$  inch) in diameter. In the left lateral ventricle anterior to the velum interpositum was a similar nodule about 3 mm. ( $\frac{1}{8}$  inch) in diameter projecting into the ventricles. Both ventricles were moderately dilated by the serous fluid. The ependyma was everywhere granular: this condition was due to small, gray, transparent tubercles. No tubercles were found in the third or fourth ventricles.

**Kidneys.**—The kidneys contained a few rather large grayish areas with here and there a yellowish speck.

The **pathological diagnosis** of the case was—

Old tubercular ulcers of the intestine,

Chronic tuberculosis of the mesenteric and bronchial lymph-glands,

Solitary tubercle of the brain,

Miliary tuberculosis of the pia, lateral ventricles, pleura, lung, spleen, kidney, peritoneum, and liver.

In connection with the other cases of tubercular meningitis which I have spoken of, I shall now mention some cases which represent the earlier periods of life, when, as I have told you, we are led to expect a variation in the symptoms and a consequent difficulty in the diagnosis. The first two cases represent tubercular meningitis as it so often appears when occurring in infants under one year.

The first case was seen by me in consultation with Dr. Kimbal, of Salem.

A male infant (Case 275), ten months old, had always been well and strong. For a few days before I saw him he had been rather dull and feverish, but had shown no other abnormal symptoms. He was evidently cutting some teeth at that time. On the day that I saw him, except that he was somewhat fretful and that he put his hands to his mouth as though his gums were disturbing him, he seemed very well, and careful physical examination revealed nothing abnormal in the ear, throat, chest, or abdomen.

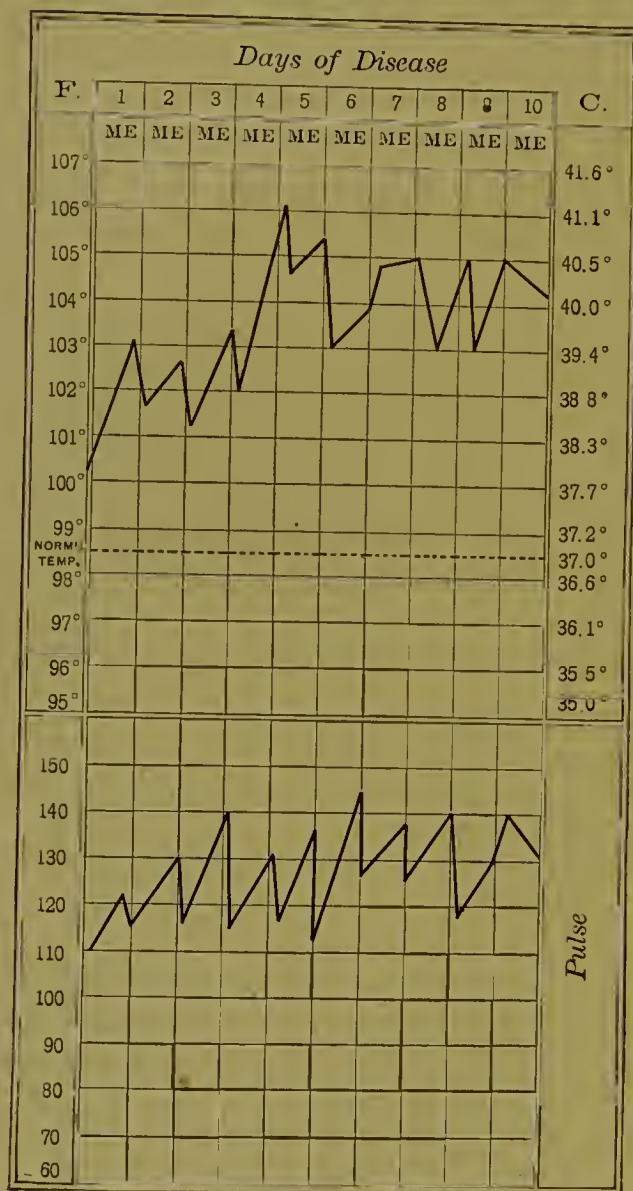


On the day following my visit the slight symptoms of indisposition which he had previously shown disappeared, and he played with a toy whistle, blowing it himself, and seeming to be very well. This condition lasted for two or three days, when he became stupid and unconscious, and about the tenth day from the time that I saw him he died in convulsions.

This case should impress upon you the difficulty of making a diagnosis in the early period of a tubercular meningitis, and how guarded we should be in giving a prognosis in young infants, even where the character of the disturbance is very slight.

The next case (Case 276) was the infant, eleven months old, whom I examined before you on the 13th of March.

CHART 23.



Tubercular meningitis. Male, 11 months old.

The history at that time was that he had always been well and strong, except that he had had bronchitis in December and that the cough had returned at intervals. He had at that time six teeth. There was a tubercular history on the mother's side of the family. He had remained well and thriving until you saw him here, when he seemed feverish and rather dull; there were anorexia and insomnia, and he was said to have become tired easily. The temperature had been rather high, 39.4° to 40° C. (103° to 104° F.), the pulse quick and regular, and the respirations rapid but regular. Nothing abnormal was found on physical examination, and, as the gums were swollen and hot, no diagnosis was given, and a guarded

prognosis. He remained in this condition until March 17, when I noticed an apparent approach to Cheyne-Stokes respiration and a little retraction of the head. There was no photophobia, and the pupils were equal and reacted well. There were no vomiting and no *tache cérébrale*. The bowels were regular; the tendon reflexes were exaggerated, but nothing else abnormal was found in the lungs, heart, or abdomen. On the following day he was found to have ptosis of the left eyelid. The fontanelle was depressed; the abdomen was distended. He gradually grew worse, and died on March 22, about ten days after pronounced symptoms of any disease had begun.

Here is the chart (Chart 23, page 624) of his temperature and pulse. The respirations during the whole course of the disease varied from 80 to 100.

This next case, which I saw in consultation with Dr. Broderick, of South Boston, represents tubercular meningitis as it appears in the second year of life. You will notice how at this period it is rapidly approaching the characteristic symptoms of the disease which are met with from the third to the seventh or eighth year, and even later.

A boy (Case 277), fifteen months old, had always been healthy. His mother was healthy, but his father had died of tuberculosis. He had twelve teeth, and was cutting one of his canine teeth, the gum over which was swollen and tender. He had always had a tendency to constipation. He was perfectly well until he was fourteen and a half months old, when he did not have a movement of the bowels for a week. He became fretful, and towards the end of the week his respiration was noticed to be of the Cheyne-Stokes type. His pulse varied from 80 to 150 and was regular. The temperature in his axilla varied from 37.4° to 38.1° C. (99.6° to 100.6° F.). At times there was rigidity of the hands, but there were no regular convulsions. He vomited at the beginning of the attack, but not afterwards.

When I saw him, in the second week of the disease, his eyes were rolling from side to side and there was much Meibomian secretion. He was unconscious, but he was said in the night to have put his hand to his head and to have cried out as though he were in pain. There was some stiffness of the neck and back. There was a rather marked *tache cérébrale*, and there was decided depression of the abdomen. The pupils were equally dilated and responded to light. There was considerable emaciation.

During the following week at one time for a few seconds he had decided opisthotonos. He gradually sank and died. The duration of the disease was four weeks.

## LECTURE XXX.

## BRAIN.—(Continued.)

## THROMBOSIS OF THE CEREBRAL SINUSES.—HYDROCEPHALUS.

**THROMBOSIS OF THE CEREBRAL SINUSES.**—Thrombosis of the cerebral sinuses is a very uncommon condition. The disease is more frequent in infancy and early childhood than in adult life. It is caused by the formation of an ante-mortem clot in one of the sinuses of the brain. As a primary condition it is exceedingly rare. It is usually secondary to some condition which has produced a deep impression upon the child's vitality, such as profound anæmia, exhausting diarrhœa, or a collection of pus in any part of the body, but especially about the scalp, as in erysipelas. A purulent otorrhœa is perhaps the most common etiological factor. It is not necessary here to do more than refer to the traumatic cases of this disease, such as involve the ear and the scalp, as in fracture, or where the disease is caused by compression, as from a cerebral tumor. The pathology of the secondary cases includes the lesions of the different processes which have caused the thrombosis. That of the idiopathic or undetermined cases is well represented in these specimens which I am about to show you (Case 279). The thrombosis may take place in any of the cerebral sinuses, and at times may occur in the course of a meningitis. When the thrombus is formed, the venous branches behind the obstruction become distended mechanically, and thus give rise to capillary hemorrhage and softening of the floor of the ventricles. When the thrombosis has taken place in the neighborhood of some inflammatory focus, such as a purulent otitis media, pyæmia may result.

So few cases have been reported where the diagnosis has been established by a post-mortem examination, that the clinical description of the disease must necessarily be very limited. The symptoms which existed in cases where this condition has been found on post-mortem examination are not such as to suffice for making a differential diagnosis during life between this and other intra-cranial conditions, such as occur in profound anæmia. Where, however, convulsions occur in an atrophic child, especially if there has been chronic trouble in the ear, we can suspect the presence of this condition after carefully differentiating all other causes. Cases of thrombosis of the lateral sinus may be suspected where symptoms of a severe purulent affection follow a suppurative otitis, with involvement of the mastoid cells, and where there is a tenderness over the external jugular vein.

The prognosis in this disease is usually fatal, except where it occurs in the lateral sinus and can be relieved by operation. Pitt reports the recovery



of a boy (Case 278) ten years old who had chronic otorrhœa, followed by acute symptoms of fever and aural tenderness. Following these symptoms, a week later, he had a rigor, and optic neuritis was developed on the right side. Exploration of the lateral sinus disclosed a clot, which was removed, and the boy recovered.

I will now show you the results of a post-mortem examination which has been made by Dr. Whitney on an infant nine weeks old.

This infant (Case 279) was seen by you with me in the wards of the Infants' Hospital two weeks ago, and at that time it was apparently well and strong. You saw it two days ago unconscious and having an irregular type of convulsions.

When first seen by me, January 16, it was, so far as I could judge, strong and healthy, weighing 4805 grammes (about  $10\frac{1}{2}$  pounds), which at six weeks is decidedly a greater weight than the average. The average weight of the male infant at birth, as I then told you, is about 3250 grammes (about 7 pounds  $2\frac{2}{3}$  ounces.) Allowing for a daily gain of 30 grammes (1 ounce), the weight of an infant six weeks old should be 4510 grammes (about 9 pounds 14 ounces), so that this infant weighed 295 grammes (about  $9\frac{5}{8}$  ounces) more than the average infant of the same age.

On entering the hospital it took its food well, had two or three apparently well-digested dejections daily, slept well, and seemed to thrive for the following week. No one would have known from its outward appearance that anything was the matter with it if it had not been carefully weighed, when it was found that it was losing. The following is the record of its weight from January 16 until its death, January 30 (Table 102) :

TABLE 102.

Date.	Weight. Grammes.	Gain or Loss. Grammes.
January 16 . . . . .	4805	
January 18 . . . . .	4655	Loss, 150
January 20 . . . . .	4630	Loss, 25
January 21 . . . . .	4595	Loss, 35
January 22 . . . . .	4610	Gain, 15
January 23 . . . . .	4590	Loss, 20
January 24 . . . . .	4425	Loss, 65
January 25 . . . . .	4420	Loss, 5
January 26 . . . . .	4420	Loss, 0
January 27, 8.30 A.M. . . . .	4110	Loss, 310
January 27, 6 P.M. . . . .	3995	Loss, 115
January 27, 7 P.M. . . . .	3925	Loss, 70
January 28 . . . . .	3945	Gain, 20
January 29 . . . . .	3965	Gain, 20
January 30 . . . . .	3735	Loss, 230

On January 23 the loss of weight was very evident, and various changes were made in the infant's food, but with no good result, as he vomited and had thin watery discharges from the bowels.

On January 28 he seemed weak and did not look well. A wet-nurse was procured for him, but her milk did not agree with him, and in fact he became much exhausted when trying to nurse.

At 7 P.M. he was examined by Dr. Haven and myself, with the following result. His temperature was  $38^{\circ}$  C. ( $100.4^{\circ}$  F.). His respirations were 35, and were natural. The pupils were normal and reacted to light. The fontanelle was very slightly depressed. The child did not seem to be in pain. Nothing abnormal was detected in the thorax, abdomen, or throat.

On January 28 he vomited considerably during the day, and had a natural yellow

fecal dejection, but he would not take his food. His pupils were contracted equally, and he had rhythmical contractions of the arms and legs, first on one side and then on the other. Accompanying these movements was opisthotonos. The head and eyes were drawn to the right. There was no rigidity or paralysis of the legs or arms. The fontanelle was not depressed. There were rapid contractions of the eyelids, first on one side and then on the other.

On January 30 he had six rather watery fecal movements. The muscular contractions ceased, but the opisthotonos continued until just before his death, which occurred at 6.30 P.M.

You see that the face is thin and pinched; the body is small and somewhat emaciated; there is slight rigor mortis; the calvaria is removed without difficulty. On close examination, nothing abnormal is noticed on the external surface of the dura mater. In the straight sinus and in the portion of the superior longitudinal sinus immediately adjoining this is a formed red clot, slightly decolorized in parts, but easily removed from the vessels, and evidently ante-mortem. The other sinuses contain a little loosely-clotted blood. The surface of the brain is moist, and the spaces between the convolutions are slightly opaque and cloudy from the presence of a serous fluid. The blood-vessels of the pia mater are injected. Upon opening the lateral ventricles and turning back the fornix the floor of the ventricles is seen to be covered with numerous thromboses of the blood-vessels and its surface to be universally reddened. The ependyma is roughened and infiltrated, and there is a bloody serous fluid in the cavity of the ventricles. The veins of the choroid plexus are filled with dark clotted blood which is directly continuous with that found in the straight sinus. The substance of the brain is moist. The spinal cord presents a moderate injection of the vessels of the pia mater. Both sides of the heart contain dark loose clots, and the heart itself is normal. The lungs are slightly œdematous. The other organs present nothing abnormal.

As a summary of the case we have an infant nine weeks old, apparently strong and well up to January 16, when it began to lose in weight. By January 23 it had lost over 200 grammes ( $6\frac{2}{3}$  ounces) without showing any other symptom of disease. By January 27 it had lost 480 grammes (16 ounces). Two days later it was attacked with convulsions and died. The autopsy showed nothing abnormal except a capillary hemorrhage into the ventricles caused by a thrombosis of the straight cerebral sinus.

The pathological diagnosis in this case is that of a sinus-thrombosis of undetermined origin, a condition which is exceedingly rare, and instances of which established by autopsy have seldom been reported.

Tirard reports the case of a boy four years of age, which is of considerable interest and value as representing secondary sinus-thrombosis.

The child (Case 280) was well until he had measles; from that time he lost in weight and strength. Just previous to coming under medical observation he had several severe convulsions, had been stupid, and had not spoken to any one.

On examination he was found to be emaciated and to have a coated tongue; his teeth were covered with sordes; his bowels were constipated. He was semi-conscious occasionally, and had slight convulsions, in which the left arm was generally affected. There was no drawing of the face; the pupils were equal; there was no strabismus, no retraction of the head, no tenderness of the spine. The patellar and plantar reflexes were present, equal and normal. There was no anæsthesia nor analgesia; a *tache cérébrale* could be obtained. There was a purulent discharge from the left ear. The urine contained a trace of albumin.

In the next two days there were several convulsions and a rise of temperature, followed by a brief return of consciousness. Examination of the chest showed dulness and crepitation over the base of the left lung.

A week later the mouth was noticed to be drawn to the left. Trembling of the hand, resembling the oscillations of paralysis rather than the movements of chorea, then appeared. When the child was lying undisturbed these tremulous motions ceased, but they became



exaggerated when the limb was raised, and were then accompanied by tremulous movements of the face. Death occurred two weeks later.

The post-mortem examination showed thrombosis of the cerebral sinuses. There were numerous small abscesses in the lungs, apparently from infarctions. The longitudinal and lateral sinuses contained well-marked deecolorized thrombi. In the latter they were soft; in the former, firm. There was pus in the left tympanum and in the mastoid sinuses. There was no perforation of the membrana tympani, and no necrosis of the petrous bone.

**HYDROCEPHALUS.**—I shall next describe a disease of the brain which is characterized primarily by an exudation of fluid into the membranes of the brain or one of its cavities.

For lack of a better name, we designate the disease by the term representing the most prominent pathological condition,—namely, hydrocephalus (water in the head).

In order that you may clearly understand what I am about to say, I shall ask you to refer again to this diagram (Diagram 8, page 594), showing a section of the skull, the cerebral membranes, and the brain.

The general shape and circumference of the head in infancy and childhood vary in the individual to a considerable degree. This has already been spoken of in a previous lecture (Division II., Lecture III., page 61), and is merely referred to here for the purpose of illustration, because the skull and its contents have so close a connection in the mind of the student.

On the other hand, when these variations in size pass a certain limit, or are combined with certain nervous phenomena, they have a distinct pathological significance.

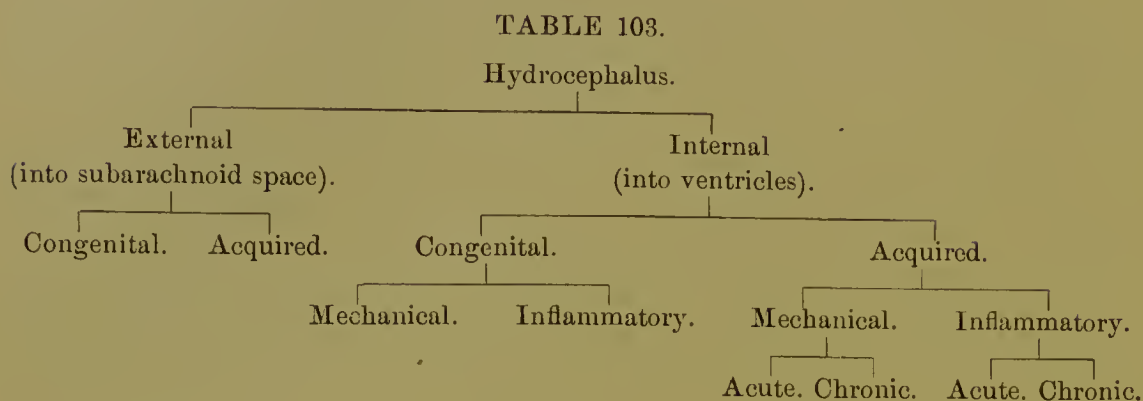
Hydrocephalus may be (1) external or (2) internal.

(1) The external variety consists in a transudation into the subarachnoid space and the meshes of the pia, represented in this diagram by Sub. A. S. (Diagram 8, page 594). This external variety is very rare, and may be either congenital or acquired.

(2) The internal and common variety of hydrocephalus consists in a transudation into the cerebral ventricles. It may be congenital (intra-uterine) or acquired (extra-uterine), and its cause may be mechanical or inflammatory. Acquired internal hydrocephalus may be acute or chronic. The acute form of the disease usually occurs as a symptom or a cause of symptoms in the course of such diseases as rhachitis, cardiac and renal disease, pertussis, and meningitis, and in various other diseases. It may be also apparently idiopathic. The chronic form of acquired internal hydrocephalus resembles so closely congenital internal hydrocephalus that we can consider them together, and, so far as the name of the disease is concerned, the term hydrocephalus would be restricted best to (1) congenital internal hydrocephalus and (2) chronic acquired internal hydrocephalus. In other words, there exists pathologically a certain class of effusions into the ventricles for which no cause is apparent. When these effusions reach a certain amount the resulting symptoms are quite typical of what is called hydrocephalus, and clinically the term has therefore been confined to cases of this class.



This table (Table 103) will aid you in understanding the classification which I have just given you :



In addition to the effusion which takes place in either external or internal hydrocephalus, there may be a combination of both, as there is a communication between the fourth ventricle and the subarachnoid space by means of the foramen of Magendie.

(1) **EXTERNAL HYDROCEPHALUS.**—External hydrocephalus may occur as a *congenital* disease, but this occurrence is so rare that little can be said concerning it. I have met, however, with one instance of a somewhat analogous condition which I saw in consultation with Dr. Broughton, of Jamaica Plain, from whom I received the complete notes of the case. It seems very likely that this was a case of congenital cyst.

A female infant (Case 281) was born of a healthy primipara, the delivery being assisted by forceps. The infant appeared to be strong and vigorous and was not cyanotic. Its head was natural in shape and size, and there was no evidence of undue or prolonged instrumental pressure. Its weight was 3632 grammes (8 pounds). Nothing unnatural was noticed about the infant for several days, except that it did not nurse well. At these times it would cry and refuse to nurse. Its mother had a sufficient supply of good breast-milk.

When it was five days old it looked rather pale and thin, and on the sixth day, when the nurse was giving it its bath, she noticed that its right arm twitched convulsively several times. This twitching increased in frequency and force and was accompanied by a marked change in the infant's face. It became very pale and was cyanotic around the lips and eyes. At times it would cry out sharply both during the convulsive twitchings and in the intervals. Sometimes it would pass into a state of semi-collapse and would be cold and very pale. At this time also it would jerk its right arm convulsively at the rate of 75 times a minute. These spasmodic movements seemed to be confined to the right arm. There was no muscular contraction elsewhere, no frothing at the mouth, unconsciousness, or other evidence of general convulsions. Sometimes the respirations would be very faint and scarcely perceptible. The pulse would be weak, about 90 per minute, and then the infant would suddenly begin to breathe with great rapidity and the pulse would increase to 120. The area of cardiac dulness was not increased. There was a moderately loud, double cardiac souffle, most marked at the second left intercostal space, but heard all over the upper part of the sternum. In the right back, at the angle of the scapula, there was a patch of dulness about 2.5 cm. (1 inch) in diameter, but there were no râles over this area of dulness. The expansion of the lungs was irregular. The temperature was slightly subnormal.

The infant was wrapped in cotton-wool and surrounded with heaters. Brandy and aromatic spirit of ammonia were given in alternate doses of 0.3 c.c. (5 minims).

On the eighth day it was reported that the convulsive twitchings had continued, and

that there had been twenty-five spasmodic attacks within the previous twenty-four hours. The infant was still in a state of collapse, the pulse and respirations were very weak and irregular, and sometimes it would actually stop breathing for a minute. It was semi-unconscious. Its pupils were dilated. The abnormal signs found in the chest were unchanged.

On the ninth day the convulsive movements had ceased, but the child was still in a state of collapse and remained perfectly dormant and passive.

On the tenth day the convulsive movements began again, and at times the infant appeared lifeless. On this day oxygen was administered for five minutes every hour. The brandy was increased to 0.72 c.c. (12 minims). The infant had been too weak to nurse for several days, and the mother's milk was given to it by means of a dropper.

On the eleventh day the oxygen was given for ten minutes at a time every hour, and brandy whenever signs of unconsciousness appeared.

During the next few days the infant began to show signs of improvement. The cardiac souffle became less distinct. The atelectasis of the lung remained unchanged. During the time when the infant was so ill there was no disturbance of the bowels or kidneys.

On the seventeenth day modified milk was substituted for the breast-milk. From this day the infant rapidly improved. The cardiac souffle lasted for six weeks, and the atelectasis gradually disappeared, the last signs of it being a little diminished respiration in the right back. The oxygen was continued in small doses for six weeks; 8400 litres (2000 gallons) were used. Of this, of course, a certain quantity was not inhaled, but escaped, as the funnel was held rather lightly over the infant's mouth. The infant had become considerably emaciated, but when it was four weeks old it had greatly improved, and, although weighing only 3405 grammes ( $7\frac{1}{2}$  pounds), looked fairly well.

When the infant was a little more than four weeks old the head was perfectly normal in shape and size. The fontanelle could be seen pulsating naturally and was normal in shape and size.

I have here a tracing (Case 281, I.) from a photograph which was taken of the infant at this time.

On the following day the head was found to be noticeably altered in shape, and this second tracing (Case 281, II.) was made from a photograph which was taken when the

## CASE 281.

I.



II.



Congenital external hydrocephalus or congenital cyst.

child was somewhat older. The rounded, full forehead had disappeared, and, instead of looking natural, the child had the appearance of an idiot. The report of the nurse was that while it was being dressed in the morning it had vomited some fluid like water, and that the head had assumed this shape within the course of a few minutes. Both segments of the frontal and the parietal bones had flattened, and apparently had settled or collapsed. The anterior fontanelle had entirely closed, and the frontal suture could not be felt. A line drawn from the vertex to the root of the nose was entirely straight, instead of showing the normal curve. The width of the forehead was also diminished. The entire frontal bone was so flat that it lay upon a lower plane than the parietal bones,—perhaps 1 cm. ( $\frac{3}{8}$  inch)



below them. The edge of the parietal bones could be plainly felt along the coronal suture, and the little finger could almost be laid upon the frontal bone in the depression. The posterior part of the head appeared to be unchanged in shape, but the skull, instead of being round and normal, had become microcephalic. The infant in other respects seemed to be in good health, took her milk naturally, and no new pathological signs were discovered.

When the infant was six months old the circumference of the head was 35 cm. ( $13\frac{1}{2}$  inches). When it was fourteen months old the head measured 36.5 cm. ( $14\frac{1}{2}$  inches). From the occiput to the root of the nose it measured 19 cm. ( $7\frac{1}{2}$  inches), from the occiput to the chin it measured 37.5 cm. ( $14\frac{3}{4}$  inches). There was a complete closure of the sutures and of the fontanelles. The infant weighed 7718 grammes (17 pounds), and was 71 cm. (2 feet 4 inches) in height. There were but few signs of intelligence. It recognized no one. It was as contented with strangers as with its mother. It was partially blind, and did not notice objects or persons, although it appeared to notice light slightly.

An examination of the eyes by Dr. Dixon showed that externally they appeared to be normal. The pupils were smaller than natural, and responded slowly to changes of light. Light was noticed somewhat, but the infant would not follow it, and it was found that it could see better from the side. The macula and disk showed no indications of inflammation or exudation. There was a very slight degree of astigmatism.

The infant could neither talk nor walk. It had a vacant manner, cried hysterically, and it sometimes required an effort to stop the crying. Otherwise it was pretty well developed. It had one tooth. The hearing was defective. There was at times digestive disturbance.

For the past three or four months there had been a return of the spasmodic twitchings of the right arm similar to those which occurred during the acute attack of ateleetasis and cardiac disturbance. During one of its digestive attacks the infant apparently had an epileptiform convulsion.

A rapid loss of cerebro-spinal fluid is not unknown, but in these cases there has usually been a history of injury. Where we do not have a history of injury we almost always find that there is a considerable amount of fluid coming from the nose, ears, or elsewhere. According to Dr. Bullard, in this case the infant seems to have first swallowed the fluid and then vomited it. The means of exit of the fluid from the skull was probably through some congenital defect at the base of the skull.

It is known that in a great many children who have hydrocephalus and similar conditions the atrophy or non-development of the brain may not show any symptoms until they are a year or more old. The parents do not notice anything, and the physician is unable to, because he has not the opportunity for sufficient observation. To determine imbecility in very young children, unless it is marked, is a very difficult matter, and even when the child is brought to the physician to determine this condition it is often impossible to decide before the second year of life, so that the fact that nothing was noticed in this especial child's (Case 281) mental condition previous to the collapse of the skull would afford no proof that there was not or had not been previously hydrocephalus, and perhaps atrophy or non-formation of a portion of the brain. It also would not be necessary in the case of congenital atrophy or non-development of the brain to have any motor paralysis or sensory disturbance, or convulsive phenomena of any kind whatever, although these symptoms usually occur under these conditions. In these cases we often find optic atrophy, but in a number of such cases no

optic atrophy can be found by means of the ophthalmoscope. In a considerable proportion of cases of this kind there is a diminution of vision which is not explained by anything that the oculists tell us.

Henoch mentions a case of hydrocephalus in which the fluid drained through the nose to the amount of 100 to 200 c.c. ( $3\frac{1}{2}$  to  $6\frac{2}{3}$  ounces) a day for quite a long time, so that the hydrocephalus was reduced considerably.

The *acquired* form of external hydrocephalus is exceedingly rare, and is usually found in connection with cerebral atrophy (hydrocephalus ex vacuo). There are certain cases which can for the present be classed under this heading until our knowledge derived from post-mortem examinations becomes more precise. These cases are so rare that it is impossible at present to formulate in detail their symptomatology and diagnosis. I have met with a few cases, however, which in their symptoms were so significant of a rapid development, with its speedily fatal issue, of an external hydrocephalus, that the diagnosis of hydrocephalus by the elimination of other possible causes has seemed to me rational, and has been supported by the post-mortem examination. The symptoms may develop, according to my experience, in young infants who either have been fairly well or have been atrophic. Physical examination in these cases has revealed nothing abnormal about the head or any of the organs, such as the heart. The infant, after a short period of indefinite symptoms, at times lasting only a few minutes, and represented by nervous twitching, perhaps a convulsion and rapid collapse, suddenly dies. I have met with three cases in my personal practice. Two were, after minute post-mortem examination by Dr. William F. Whitney, found to represent as their only pathological lesion external hydrocephalus with œdema of the cerebral substance. The third case showed this condition merely as a symptom of pernicious anæmia, and I have spoken of it in a previous lecture.

One of these cases was an infant (Case 282), ten months old, of healthy parentage, and always perfectly well, except that for two weeks before its death it had cried more than usual and was somewhat irritable. Five days before its death it was somewhat languid, but took its food well, and when I examined it the night before its death nothing abnormal was found. On the following morning it had a few convulsive movements and died suddenly.

On post-mortem examination nothing abnormal was found, except that a large amount of cerebro-spinal fluid escaped from the cranium as soon as the skull and parietal dura mater were removed. There was also a general œdematous condition of the brain.

The other case was a female infant (Case 283), seven and one-half months old, which had been suffering from malnutrition for several months and was very weak and puny. On the day of its death I examined it carefully, and, with the exception of an atrophied condition of the muscles and a weak action of the heart, nothing abnormal was discovered. Within an hour after I had seen the infant it had a few convulsive attacks and died suddenly.

The autopsy, made twenty-four hours after death, showed nothing abnormal externally. Rigor mortis was present. There was great pallor of all the organs. The skull was normal in development, and the fontanelle was normal. There was cerebro-spinal fluid in excess. The brain-substance was very moist and pale, but otherwise nothing abnormal was noticed in the brain or meninges. The heart was of normal size, and its cavities and valves were



normal. The ductus arteriosus and Eustachian valve were closed. The muscular substance on the right side of the heart was pale and opaque, while that of the papillary muscles on the left side was pale but not opaque. Microscopic examination showed the muscular substance to be filled with minute, highly refracting granules, which in part dissolved on the addition of acetic acid, but were left undissolved in some places, where the structure of the fibre was destroyed. On the left side of the heart granules were present which could be wholly dissolved in the acetic acid. There was fatty degeneration more or less marked of the cardiac muscles and also of the diaphragm, the fibres of which showed numerous granules, which dissolved in acetic acid in about one fibre to fifty. The kidneys were pale, but otherwise nothing abnormal, either microscopically or macroscopically, was found. The supra-renal capsules were normal. The liver was of normal size, and on section its surface was found to be dry, yellowish, and opaque. Microscopic examination showed that the liver-cells were filled with fat-drops of varying size, especially numerous in the cells of the periphery of the lobule. The cells themselves had a sharp outline and a well-defined nucleus. The pancreas was normal. The stomach was normal in size, and contained considerable milk, with but few curds. Nothing abnormal was detected in it by microscopic examination. The small intestine contained a very little soft, yellowish material. The large intestine contained a small amount of yellowish, soft faeces. No enlargement of Peyer's patches or of the solitary follicles was found. The mucous coat was normal.

(2) **INTERNAL HYDROCEPHALUS.**—Internal hydrocephalus may be congenital or acquired. The earlier the hydrocephalic condition begins, the larger will the cranium become. We therefore find the very large heads, as a rule, to be of the congenital variety. The head is at times of such a size as to cause difficulty in the delivery, or the fluid may collect very rapidly after birth, and the head soon assumes the characteristic appearance of hydrocephalus.

This skull of a child three years old (Fig. 92) is an exaggerated type of the congenital internal hydrocephalic head.

This other skull of a child, also three years old (Fig. 93), which I place beside the hydrocephalic skull, represents a normal head of the same age.

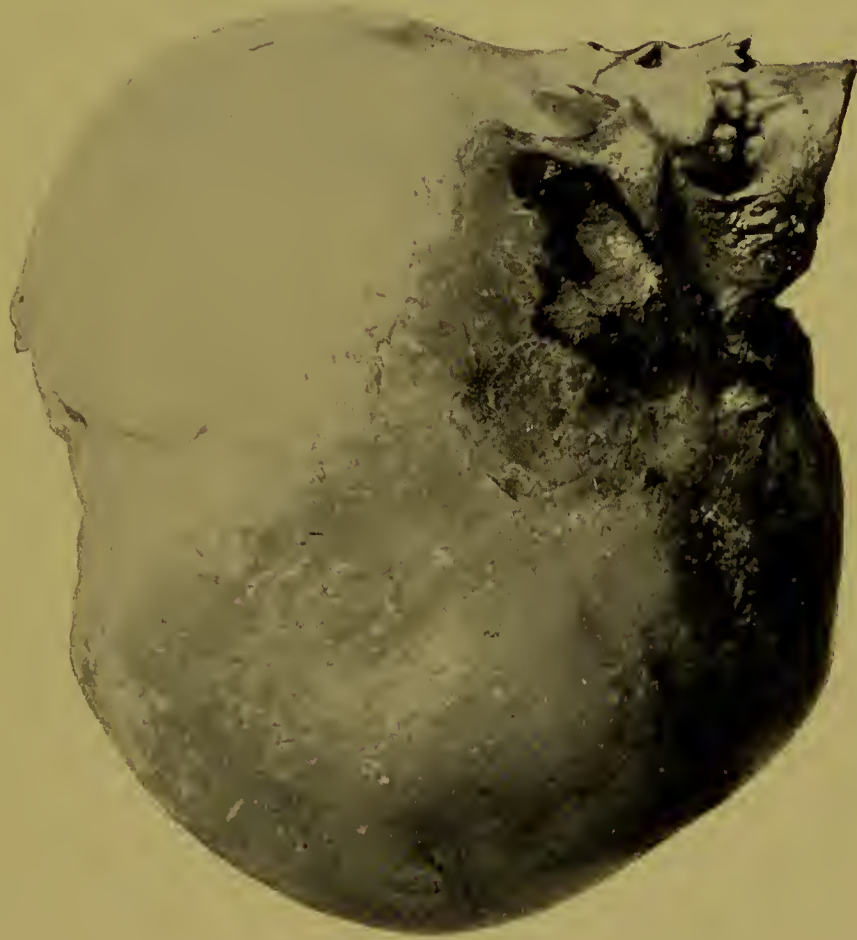
The face in these cases of hydrocephalus remains about the same size as it would be normally, but usually looks much smaller from the disproportionate size of the head, which rests upon it from above like a globe.

**Congenital Internal Hydrocephalus.**—The cause of congenital internal hydrocephalus is somewhat obscure. In some cases it is of inflammatory origin, in others no evidence of inflammation can be found.

**PATHOLOGY.**—The anatomical appearance of the brain itself, as a rule, corresponds with and may be accepted as the result of pressure by an intra-ventricular fluid. This brain (Fig. 94, page 635) was taken from a child who died of congenital internal hydrocephalus, and well exemplifies the pathology of the disease.

You see that the convolutions are flattened and that the walls of the ventricles are much thinned by the intra-ventricular pressure, while the ventricles themselves are much dilated. In some parts the cortex is less than 1 cm. ( $\frac{3}{8}$  inch) in thickness. The amount of fluid in these cases varies from a few cubic centimetres to three or four litres. The fluid has a specific gravity of about 1004.

FIG. 92.



Hydrocephalic skull, child 3 years.

Warren Museum, Harvard University.

FIG. 93.



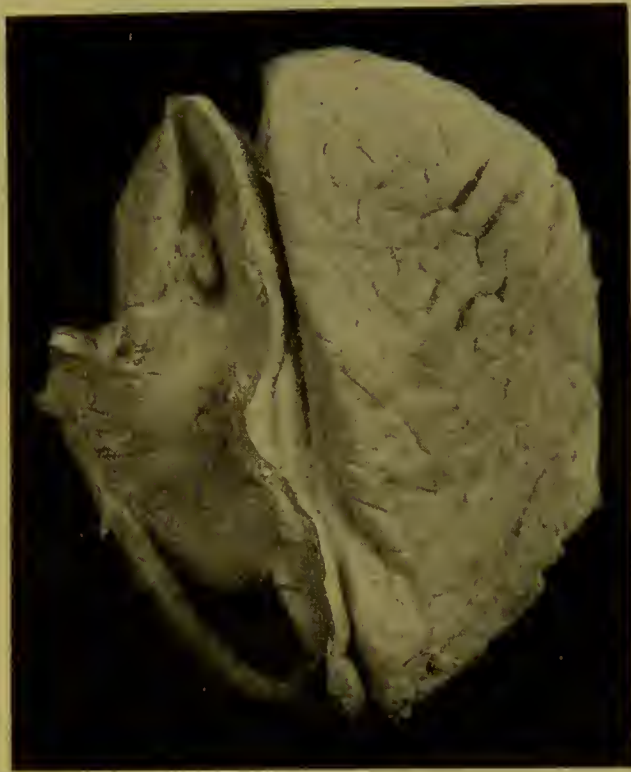
Normal skull, child 3 years.





**SYMPTOMS.**—The symptoms of congenital internal hydrocephalus are essentially those caused by pressure. We naturally, therefore, find the fontanelles bulging and fluctuating, and the bones thin and forced out of position. As you see in this skull (Fig. 92), the temporal and parietal bones diverge as they extend upward, while in the normal skull they ascend almost perpendicularly. If the disease has existed for some time, the upper wall of the orbit becomes flat and the eyeballs protrude. The

FIG. 94.



Hydrocephalic brain. Warren Museum. Harvard University.

intra-cerebral pressure often produces a strong collateral circulation in the scalp and the forehead, where the vessels appear like tortuous blue cords. Functional disturbances are numerous, and vary in almost every case. As a rule, the children are idiotic, but at times, even in marked hydrocephalus, we find the mental condition normal, even when paralysis is present. A notable instance of this fact is presented by this little girl whom I have had brought here to-day to show you.

This child (Case 284, page 636) is five years old.

You see that, although she has completely lost the power of using her legs, and has a large head and distended, bulging fontanelle, yet she is bright and intelligent. She was nursed by her mother for over a year, and cut her first tooth when she was six months old. She was always well and strong, but high-tempered. She has never had any disease. When she was five months old she fell from her crib and struck her head, but it did not seem to hurt her especially. Her head was always noticed to be of a peculiar shape. When she was eight months old she fell out of a chair and was stunned, but was not otherwise hurt. She has had convulsions from time to time, but her mind has always been bright. She was unable to hold up her head until she was three years old, and has always complained of more or less frontal headache. Her appetite has always been excessive, and



her taste for food somewhat peculiar. She has gradually grown stronger, and is beginning to attempt to walk. She sleeps well, and her bowels are regular. Her head measures 57 cm. (22½ inches).

CASE 284.



Congenital internal hydrocephalus. Female, 5 years old.

As the various cerebral centres become affected by pressure, we notice that symptoms arise corresponding to the parts of the brain which are affected. Among these symptoms are nystagmus and, less frequently, strabismus. The pupils at first are usually moderately dilated. Later they become fixed, and sensibility to light is lost. The hearing lasts for a long time. The ability to walk is interfered with. Partial or general convulsions, paralysis (usually paraplegic), and contractures may occur. Pain in the head is often complained of, but, as a rule, is not so severe as in meningitis. There is difficulty in keeping the head erect, as it is so heavy. The digestion is often good, and the appetite usually extreme. The respiration is normal from adaptation. The pulse is usually not retarded. The temperature, as a rule, is normal. The adipose tissue is often abnormally increased.

**DIAGNOSIS.**—As congenital internal hydrocephalus is almost invariably attended by enlargement of the head and separation of the sutures, the diagnosis is not especially difficult, and is determined by comparing the measurements of the head with those of a normal head of the same age. I have described the measurements of a normal head in a previous lecture (Lecture III., page 61). In addition to the enlargement of the head, the symptoms of direct intra-cephalic pressure make the diagnosis very simple.

**PROGNOSIS.**—These congenital cases, as a rule, die before childhood has been reached, but they have been known to live to middle age. Death usually occurs from some intercurrent affection. Complete recovery is very rare.

**TREATMENT.**—The treatment of congenital internal hydrocephalus has been varied, but without marked success. When the effusion is not large and is not increasing, moderate pressure with a rubber bandage seems to have a favorable result. Where the disease is apparently not in an active state and is characterized by a very slight increase of fluid, aspiration

through the anterior fontanelle of a small quantity of fluid at a time has been of temporary benefit. The point of aspiration should be 2 to 3 cm. ( $\frac{5}{8}$  inch to  $1\frac{1}{4}$  inches) from the median line, so as to avoid puncturing the longitudinal sinus. In this way the condition of the patient is often rendered more satisfactory.

An operation for chronic hydrocephalus presents no technical difficulties. Of course only certain cases are suitable for operation. Moderate effusions should be let alone, also those cases where a rudimentary development of the brain is suspected. Cases where an operation is especially indicated are comparatively both physically and mentally well developed up to the time when the enlargement of the cranium began. Such children should show the symptoms of direct intra-cranial pressure. They are evidently becoming weak-minded or idiotic. They do not learn to talk, or they quickly forget what they have learned. They may also have become totally blind. The power of walking is interfered with. Contractions and partial and general spasms are of ordinary occurrence. Unless the pressure is speedily removed, atrophy of the brain results, and if they live they remain idiots for life. Such cases as these you can best refer to those who are skilled in neurology and surgery.

I have in my wards to-day a number of cases of congenital internal hydrocephalus to show to you, which are of considerable interest in view of what I have just told you.

Of these illustrative cases I will first show you this infant (Case 285), two years old, which is sitting in its mother's lap.

The special point of interest in this case is that the circumference of the mother's head and that of the child's head are almost identical, 52 cm. ( $20\frac{1}{2}$  inches). You will notice the overhanging brow and deep-set eyes, the globe-shaped head and open bulging fontanelles, the small face and oblique parietal bones of the infant's head in comparison with the normal, round shape of the mother's head.

The history, so far as the mother is concerned, is negative. She has had no miscarriages. The infant was born at term, and cut its first tooth when it was six months old. It now has sixteen teeth. It has had no convulsions. It has for some time supported its head alone and sits alone, but has never attempted to walk. The intelligence seems normal. It is apt to sleep with its eyelids partly open. Its digestion is good, and its appetite is very good. The bowels are rather relaxed. On examining the head you will see that it is abnormally large. From the root of the nose to the occipital prominence it measures 32 cm. ( $12\frac{1}{2}$  inches). From the base of one mastoid to that of the other it measures 33.5 cm. ( $13\frac{1}{2}$  inches). The position and movements of the eyes are normal. You will notice, on looking at the head from above, that it is triangular in shape, with the base of the triangle at the occiput. The anterior fontanelle you see is widely open, and is about 4 cm. ( $1\frac{1}{4}$  inches) in width and length. The protruding overhanging forehead makes the face look small. The epiphyses are not enlarged. Examination of the lungs, heart, and spleen shows nothing abnormal. The child weighs 10,442 grammes (23 pounds).

In this next bed is a boy (Case 286, page 638), three and a half years old, whose head is typical of congenital internal hydrocephalus. The circumference of the head is 57 cm. ( $22\frac{1}{2}$  inches).

There is no history of disease in the parents, and the mother has had no miscarriages. The child's head has always been large since birth. He has never had any convulsions. He cut two teeth when he was four months old, and when he was a year old he had ten teeth. He walked when he was fourteen months old, but his legs never seemed strong.

He has never had any paralysis, but he gets tired easily. Nine months ago he fell down one step, and half an hour later began to vomit and was somnolent. During the following

I.

CASE 286.

II.



Congenital internal hydrocephalus. Male,  $3\frac{1}{2}$  years old.

two days the vomiting and somnolence continued, but he was never unconscious. His head then began to increase in size, so that his mother had to buy him larger hats. Before the accident he had always held his head up. He talked when he was fifteen months old, and

CASE 287.



Congenital internal hydrocephalus.

seemed to be an unusually bright child. He holds his eyelids partly open when he is asleep, and he has lately had strabismus of one of his eyes when he looks steadily at an object. The head is markedly enlarged, with a broad, protruding fontanelle, and is rather flattened at



the vertex. The superficial veins of the head are prominent. The face, as in the other child (Case 285), is small. The movements of the eyes are normal. From the tip of one mastoid process to that of the other is 41.5 cm. ( $16\frac{1}{4}$  inches). From the base of the nose to the occipital protuberance is also 41.5 cm. ( $16\frac{1}{4}$  inches). The anterior fontanelle is widely open and is 2.5 cm. (1 inch) long and 2.5 cm. (1 inch) wide. An examination of the heart, lungs, liver, and spleen shows that they are normal. The abdomen is prominent. The radial epiphyses are enlarged, and there is a slight outward bowing of each tibia. The spine is straight. The patellar reflexes are not increased, and there is no ankle-clonus. The urine is pale, thin, and clear, and contains no albumin. An examination of the eyes by Dr. Davis shows no marked diminution of vision in either eye. They are hypermetropic, and there is a convergent strabismus, probably accommodative. The optic disks are rather wider than usual, and their vessels diminished in size. There are no other signs of optic atrophy. There is no dilatation of the retinal veins or swelling of the disks. The examination, therefore, shows that, with the exception of an early stage of atrophy of the optic nerve from pressure, the fundus oculi is negative.

In this case a chronic congenital effusion was apparently actively increased by a blow on the head.

This little boy (Case 287, page 638), a patient of Dr. Haven's, is an interesting case of hydrocephalus, with its accompanying disturbance of the motor function of the legs, and also mental impairment. He is a characteristic picture of the disease. He cannot walk,

## CASE 288.

I.



II.



Internal hydrocephalus (probably congenital). Female, 6 years old.

but is able to sit in a chair. His legs are atrophic, his abdomen is distended, and he is somewhat emaciated. His head, as you see, is decidedly enlarged, and he is mentally weak. His appetite is excessive. He is very fretful and peevish.

Cases of this kind are very apt to live for only a few years, and are especially liable to die if they are attacked by any intercurrent disease, such as pertussis.

This little girl (Case 288) is six years old.

She is said to have been normally developed and healthy at birth, but was unable to hold her head up until she was two years old. She has never walked.

You notice on looking at the head, both in front and in profile, that it is abnormally large. It measures 65 cm. (26 inches). The movements of the hands and arms are normal. She cannot stand unless she is supported, and there is a spastic condition of the legs, with an exaggeration of the knee-jerks. She articulates well.

She represents a case of partial recovery from chronic hydrocephalus, probably of the congenital variety. Her general development will probably always be interfered with.

I shall now ask you to come to the operating-room and see some cases of chronic congenital internal hydrocephalus which Dr. Lovett is about to operate upon.

This first infant (Case 289, I.) is six months old.

It was noticed when the child was one week old that its head was beginning to increase in size. When two months old the circumference of the head is said to have been 41.5 cm. (16½ inches). Somewhat later the circumference of the head was 44 cm. (17¼ inches),

#### CASE 289.

##### I.



Congenital internal hydrocephalus. Male, 6 months old.

and when it was three months old the circumference was 45.5 cm. (18 inches). When it was five months old the circumference was 55 cm. (21¾ inches). To-day, as you see, it measures 57.5 cm. (22¾ inches). There is no history of syphilis or of tuberculosis in the family. The infant has had no marked convulsions, although some twitchings of the hands and feet have been noticed. There has been constant nystagmus, and the infant's general condition is atrophic. You will notice the marked prominence of the eyes, and the great distention of the head. The anterior fontanelle is very large, and the skin covering it is distended to such a degree that it is thin and glistening. There is no doubt in a case of this kind that aspiration of the cerebro-spinal fluid should be made for the purpose of relieving the general condition.

Dr. Lovett, as you see, has just made an exploratory puncture at the vertex of the right side of the head, about 5 cm. (2 inches) from the median line. In place of the small trocar he now introduces a larger one. Through this large trocar he has passed several strands of silk to serve as a drainage by capillary attraction. You will now notice (Case 289, II.) after withdrawing the fluid from the right ventricle that the right parietal bone has sunk in, its edge being beneath that of the left parietal bone, which is still pushed outward by the fluid in the left ventricle. On measuring, a considerable quantity of fluid is found to have been aspirated.

You see that, although the head is very much reduced in size, there are no symptoms of collapse nor any other alarming symptoms shown by the infant.

CASE 289.

II.



Congenital internal hydrocephalus after aspiration of right ventricle.

III.



Congenital internal hydrocephalus after aspiration of both ventricles.





(Subsequent history.) The infant was very restless during the following night, tossing its head about and crying.

On the next day 270 c.c. (9 ounces) of clear fluid were withdrawn from the left ventricle by introducing the trocar at a point corresponding to the point of aspiration of the right ventricle. The head was then found to measure 55.5 cm. (22 inches).

A No. 8 soft catheter was then introduced into each ventricle and sewed into place. The external end of each catheter was closed by bending it upon itself and tying it tightly with a silk ligature.

The appearance of the cranium after the second aspiration is here shown (Case 289, III.). You will notice the great depression of the anterior fontanelle.

Three days later 120 c.c. (4 ounces) of fluid were drawn through the catheter.

On the following day 138 c.c. ( $4\frac{3}{5}$  ounces) of fluid were withdrawn, and the infant was found to have a better facial expression.

On the next day, the fifth after the operation, 105 c.c. ( $3\frac{1}{2}$  ounces) of fluid were removed, and the head was found to measure 51 cm. ( $20\frac{1}{4}$  inches).

On the following day Dr. Dane began a series of observations on the fluid-pressure in this case, which were the first of the kind that have been brought to my notice. He connected the catheter with a manometer and found a positive pressure of 7 cm. When the infant cried the pressure rose to 12 cm. On this day 120 c.c. (4 ounces) of fluid were removed.

On the following day the pressure was found to be 4 cm., and rose to 5 cm. when the infant cried. 90 c.c. (3 ounces) were removed.

On the following day the pressure was the same. 68 c.c. ( $2\frac{1}{4}$  ounces) of fluid were removed on this day, and the head was found to measure 49.5 cm. ( $19\frac{5}{8}$  inches).

On the following day the infant failed rapidly, had convulsions, became unconseious, and died in the evening.

After death 556 c.c. ( $18\frac{1}{2}$  ounces) of cerebro-spinal fluid were removed. The specific gravity of this fluid was 1003. It contained  $1\frac{7}{8}$  grammes (28 grains) of albumin to the litre. This was measured by an Esbaeh's albuminimeter.

The total amount of fluid withdrawn from the ventricles in this case was 720 c.c. (24 ounces) in sevenappings.

The next case is that of an infant (Case 290), seven months old, who was admitted to the hospital to-day.

It has always been nursed. When it was two days old it had convulsions. Three weeks later it had bronchitis, and accompanying this disease a return of the convulsions,

#### CASE 290.



Congenital internal hydrocephalus. Male, 7 months old.

which occurred as often as six or seven times in the day. They were localized in the left arm and left leg. These convulsions lasted for three weeks, gradually growing less severe. There was at this time a certain amount of intestinal disturbance, which, however, has now disappeared. There was also a history of a purulent discharge from the ears before the infant was admitted to the hospital. It cried out sharply at night. The measurements of the head are 56.5 cm. ( $22\frac{3}{8}$  inches) in circumference, and 36.7 cm. ( $14\frac{1}{2}$  inches) from ear to ear over the vertex. The anterior fontanelle is bulging. The eyes, as you see, are markedly divergent and protrude from the orbits. If you will observe the eyes closely you will see

that there is at times a slight trembling and twitching. No other spasmodic movements are noticed. The chest measures 33.5 cm. ( $13\frac{1}{4}$  inches) in circumference.

You see that the child as it now lies on the operating-table takes no notice of anything. It has been decided to relieve the cerebral symptoms by aspiration on account of the great increase in the intraventricular fluid shown by symptoms of increased intra-cranial pressure.

As you see, Dr. Lovett has introduced a thoroughly aseptic aspirating needle into the right lateral ventricle through the much dilated anterior fontanelle. The aspirating needle is connected with a water manometer, which shows a pressure of 30 cm. Having determined the pressure by means of this water manometer, we can now remove a certain amount of the fluid. In order to do this, a whiff of ether is given to the infant, and you see that Dr. Lovett introduces a trocar in place of the aspirating needle. He then withdraws the trocar, leaving the canula in the cavity. Next, as you see, he introduces a No. 7 soft rubber catheter through the canula, and on withdrawing the latter the end of the catheter is left in the ventricle. 130 c.c. ( $4\frac{1}{2}$  ounces) of clear fluid have been removed from the ventricle. The specific gravity of this fluid is 1006. You perceive that the axes of the eyes, which before the operation were divergent, are now parallel. The external end of the catheter is now closed in the same manner as you saw it done in the preceding case.

(Subsequent history.) The observations on the intra-cranial pressure in this case, as in the last (Case 289), were made by Dr. John Dane. On the day following the operation the pressure was found to be 14 cm. by the water manometer. When the child cried it was increased to 20 cm. 25 c.c. ( $\frac{5}{8}$  ounce) were removed on this day, and the circumference of the head was then found to be 35 cm. ( $13\frac{7}{8}$  inches). The specific gravity of this fluid was 1007.

On the second day after the operation the pressure was found to be the same. At that time 55 c.c. ( $1\frac{5}{8}$  ounces) of fluid were removed, the specific gravity of which was found to be 1006.

On the third day after the operation the tube was found to have leaked a little, and there was a slight convulsion in the morning. 50 c.c. ( $1\frac{3}{8}$  ounces) of fluid were removed.

On the following day the tube was found to be leaking freely, and the infant was in a state of collapse and refused to nurse. The head measured 52 cm. ( $20\frac{1}{2}$  inches).

On the next day there was still some leakage around the tube, but the infant was in a better condition.

Three days later, the leakage around the tube having been controlled in the mean time, the infant seemed better, but it had a thick purulent discharge from both ears.

During the next few days the child began to grow weak, and there was again a slight leakage around the tube.

On the eleventh day following the operation the child died quietly, no convulsive symptoms having appeared.

There was no complete post-mortem examination, but the distended ventricles were found to contain 759 c.c. ( $25\frac{1}{3}$  ounces) of clear straw-colored fluid,—the left ventricle containing 409 c.c. ( $13\frac{2}{3}$  ounces) and the right 350 c.c. ( $11\frac{2}{3}$  ounces). An examination of this fluid by Dr. J. H. Wright showed that it was turbid with a flaky sediment. It was slightly alkaline. The specific gravity was 1009. It contained about 0.1 per cent. of albumin. No sugar was found. Under the microscope nothing was seen resembling the lining cells of the ventricles. An inoculation of a guinea-pig with this fluid to determine whether it was of a tubercular nature or not gave negative results.

**Acquired Internal Hydrocephalus.**—Both the acquired and the congenital form of internal hydrocephalus may be of mechanical or inflammatory origin, but the acquired form shows evidence of an inflammatory condition oftener than does the congenital form, and occurs very frequently in connection with rhachitis. Acquired internal hydrocephalus may be acute or chronic. In its acute form it may occur at any age as a symptom of any



one of a number of diseases, such as meningitis, one of the exanthemata, pertussis, and rachitis. It may in any of these forms become chronic. The disease may sometimes appear to be idiopathic.

The chronic form of acquired internal hydrocephalus occurs usually in the first four years of life, and is represented pathologically by a small amount of intra-ventricular fluid, perhaps 100 or 200 c.c. ( $3\frac{1}{2}$  to  $6\frac{2}{3}$  ounces). It is this chronic form of acquired internal hydrocephalus that can best be classified under the name of hydrocephalus with the congenital internal hydrocephalic cases which I have just shown you.

**SYMPTOMS.**—The symptoms of the acute form of acquired internal hydrocephalus are so closely connected with the diseases in which it occurs as a symptom that it is not necessary to speak of them here.

The symptoms of chronic acquired internal hydrocephalus are very much the same as those of the congenital form. The firmer the union of the bones the less likely is enlargement of the head to occur.

**PROGNOSIS.**—The prognosis as regards life is serious. Of those who recover, many are left either with some mental defect or with permanent blindness, the latter the result of optic atrophy. Complete recovery may occur, but is exceedingly rare.

**DIAGNOSIS.**—The diagnosis of chronic acquired internal hydrocephalus of the idiopathic form is in its earlier manifestations chiefly made by the elimination of other cerebral diseases, though after the stage of inflammatory irritation has passed and the symptoms of pressure have become established, a provisional diagnosis can usually be made. I say provisional because the disease is rare, and a sufficient number of autopsies have not yet been made to justify a decided diagnosis such as can be made in the congenital form of the disease.

**TREATMENT.**—The treatment is purely symptomatic in cases where the sutures and fontanelles have completely closed, except where it is advisable to perform craniectomy. Where they have not closed, the treatment is the same as in the congenital form,—that is, usually operative.

I have here three cases which I feel justified in reporting to you as probably representing chronic acquired internal hydrocephalus. Of course in these cases we must allow that a tubercular or syphilitic taint may have been the starting-point of the intra-ventricular disease.

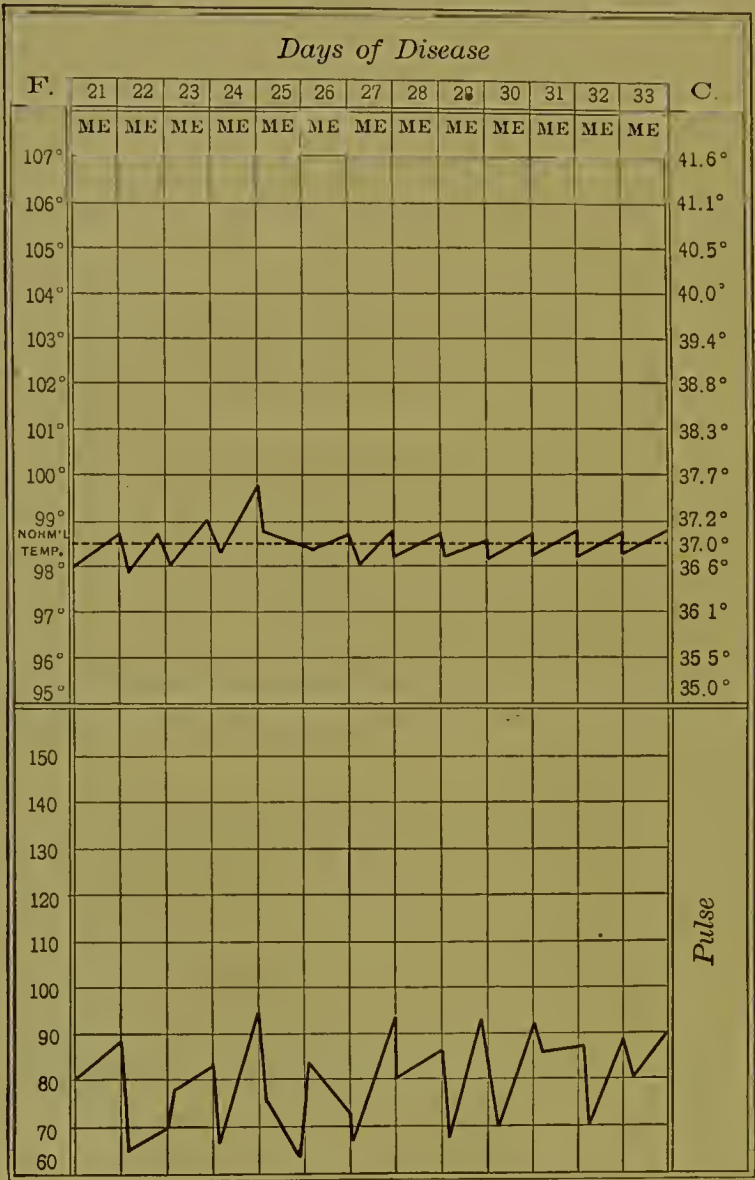
A boy (Case 291), four years and eight months old, was seen by me in consultation with Dr. E. J. Forster, May 27, 1885. The child's parents were healthy; his mother had other healthy children and had had no misadventures. The child had always been well, measles being the only disease which he had ever had. At the age of six months, while in the process of cutting a tooth, he had three convulsions, from which he recovered entirely. His appetite had always been capricious, but his digestion was good. His bowels had always been regular. He had lately come from a malarial region, where he had lived in a rather damp dwelling for a year.

On May 6 he vomited twelve or thirteen times. The vomiting then stopped, but returned later from time to time. He complained of pain in his stomach, had no fever, and sometimes appeared to feel chilly. His bowels were constipated, and in the beginning of

the attack his pulse was slow. He had been subject to night-terrors for some time previous to this sickness. The vomiting had lessened by May 27, and the report of my examination on that date is as follows:

Pulse 60, rhythmical; respirations regular; temperature normal; has had earache lately; no discharge from the ear since he was an infant; the examination of the ear was negative; yesterday morning he had a general clonic convulsion lasting for some time; his tongue is slightly coated; he lies in an apathetic state, though perfectly conscious; he is losing in weight and strength and has lost his appetite; urine normal. Nothing abnormal is found on examination of the thorax or abdomen. (The examination of the eyes, June 15,

CHART 24.



Chronic acquired internal hydrocephalus.

by Professor O. F. Wadsworth, showed that there was much swelling of the optic nerve, increased prominence of the retinal vessels, hemorrhages, and neuritis.) The child seems much brighter, and plays about. His intelligence is perfectly good; he has had no more convulsions and no paralysis, and seems perfectly well, except that his pupils are dilated and he is totally blind. The accompanying chart (Chart 24) records his temperature and pulse from May 27 to June 9.

This child came to see me in May, 1893, when he was twelve years old. He had been and was at that time perfectly well. He was a bright, well-developed, healthy boy, twelve years old. His pupils reacted, but he had never recovered his sight. He weighed 19.8

kilogrammes (90 pounds). His bowels were regular; his appetite was good. His knee-jerks were not increased. His head measured 49 cm. (19½ inches).

I shall now call your attention to this little girl (Case 292), ten years old, who has been in the hospital for about two months. Her family history is negative, with the exception that two maternal aunts died of phthisis. The child has never had any disease except bronchitis, measles, and varicella. Her present trouble began one and a half years ago with at first attacks of loss of consciousness without convulsions, lasting half an hour, after which she would fall asleep for some time. Eighteen months ago these attacks began to be accompanied by convulsions, which usually came about once a month, the intervals sometimes being three or four months. The duration of the convulsions and the following sleep were about the same as in the earlier attacks. These convulsions have now not occurred for six months, with the exception of one slight attack five weeks ago, when the right eye twitched and there was a momentary loss of consciousness. The convulsions began with twitching in the right eye, followed by twitching of the right hand. The rest of the body was not affected. The attacks were ushered in by intense headache, sudden vomiting, fever, flushed face, and retraction of the head. The headaches, which began about one year ago, were extremely severe and caused her to scream with pain. They were felt all over the head, but especially in the region of the occiput; they would last an hour or more, until she vomited and then fell asleep. They came about every day, but were not always accompanied by vomiting. The headaches ceased altogether for a time, but she has had two or three in the past five weeks. She has lately complained of dimness of vision. Six weeks ago she had pains in her right hand between the fore and middle fingers and began to lose the use of her hand. Physical examination reveals, as you see, nothing definite. She is more awkward in using her right hand than her left, but all motions, you will perceive, are possible and strong. Her right foot seems to drag a little and is a little weak after running, but these symptoms are not especially marked. The sensation of the hand is normal; the knee-jerk is somewhat increased. For the past six weeks she has shown evidence of facial paralysis. Her pupils at times have been widely dilated. Dr. Dixon reports an atrophy of both disks, with slight myopia.

#### CASE 293.

##### I.



Acquired internal hydrocephalus. Protrusion of eyes.

She has been sleeping poorly, and has had a fair appetite; the temperature has been about 37.7° to 38.3° C. (100° to 101° F.); the pulse 96 and regular; the respirations 24 and regular. She was treated with a good general diet and 0.30 gramme (5 grains) of bromide of potassium three times a day. The bromide was omitted one month ago. Lately she has seemed to be much better, and, as you see, she is now looking very well.

This little boy (Case 293, I.) is two and a half years old.



He is said to have been well and strong at birth, and never to have been sick until two months ago, when he woke up screaming in the night, and this was followed by convulsions. For two weeks he did not recognize any one, cried out at times, and micturition and defecation took place unconsciously. In the early days of the attack he lay immovable. After consciousness returned he improved for five or six weeks, and no other especial abnormal condition developed. Two weeks ago he was attacked with convulsions, occurring at intervals of from thirty-six to forty-eight hours and lasting from one to one and a half hours. These attacks were ushered in by crying, which was followed by loss of consciousness, opisthotonos, kicking, and finally clonic convulsions. His mother states that during the early weeks of the disease he shrieked at times continuously and evidently suffered the most acute pain, apparently in the head.

## CASE 293.

## II.



Acquired internal hydrocephalus. Kernig's symptom. Male,  $2\frac{1}{2}$  years old.

On examining the child, you see that he is well developed and nourished. The anterior fontanelle is still open. The fronto-parietal suture on the right side of the head is quite distended. His forehead is rather bulging. His eyes are somewhat prominent, and rather depressed in the orbits. The pupils are dilated. The head measures 47.3 cm. ( $18\frac{3}{4}$  inches) in circumference, 33.2 cm. ( $13\frac{1}{2}$  inches) from glabella toinion, 27.2 cm. ( $10\frac{3}{4}$  inches) from ear to ear. The circumference of the chest is 49.7 cm. ( $19\frac{7}{8}$  inches). There is a slight hemiplegia and paresis of the right arm and leg, but objects can be grasped with the right hand. He cannot walk. There are no enlarged glands. Nothing abnormal is found on examination of the heart, lungs, or spleen. The knee-jerks are increased, the right one more than the left. There is no ankle-clonus. The teeth are in good condition.

On placing the child on the edge of a table (Case 293, II.), you will see that both the legs become stiffened (Kernig's symptom).

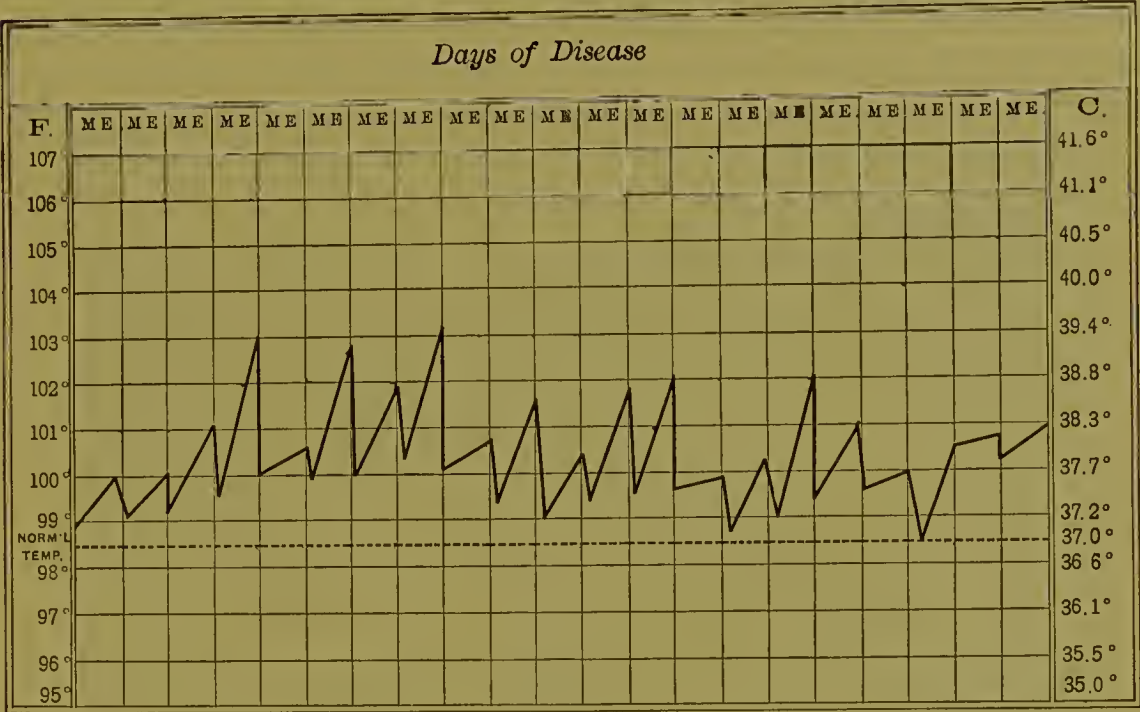
The child seems to be fairly bright and to be improving every day. He is much less fretful than formerly. An examination of the eyes by Dr. Jack shows a beginning atrophy of the optic nerves, with retinal hemorrhages of the left eye.

The child's temperature has varied usually from 37.7° to 38.3° C. (100° to 101° F.). The pulse has been regular and somewhat quickened.

The diagnosis of this case is evidently one of intra-cranial disease. The disease was acute in its onset, and was accompanied by extreme pain in the head, convulsions, and unconsciousness, followed by a partial paralysis of the arm and by loss of the power of walking. The protrusion of the eyes would indicate intra-cranial pressure, and the paralysis some intra-cranial lesion, possibly of mechanical origin. What the nature of the original attack was cannot now be determined, but it was evidently of an acute inflammatory type, and it seems as though it must have been connected with an inflammatory condition probably affecting the ventricles. Following this inflammatory condition, the symptoms indicate an intra-ventricular effusion, and I think we can therefore assume that, whatever the original cause of the disease was, the child may now be said to have chronic acquired internal hydrocephalus.

(Subsequent history.) During the following year the child improved slowly but markedly. He became less fretful; he learned to talk better, and finally to walk. His

CHART 25.



Chronic acquired internal hydrocephalus. Male, 2½ years old.

temperature became normal, and when last seen, at the age of three and a half years, he seemed to be perfectly well, the paralysis of the arm and hand having almost disappeared. Here is the temperature chart (Chart 25), showing the course of the temperature for twenty-one days in the third month following the original attack.

## LECTURE XXXI.

## BRAIN.—(Concluded.)

CEREBRAL ABSCESS.—CEREBRAL PARALYSIS.—ATHETOSIS.—INTRA-CRANIAL TUMORS.  
—INTRA-CRANIAL SYPHILIS.—IDIOCY.—MIRROR WRITING.

**CEREBRAL ABSCESS.**—Cerebral abscess is a localized purulent encephalitis. It is probably always secondary to suppurative disease elsewhere. It may arise from a suppurative condition of the scalp, but its most common source is some purulent disease of the ear or its surroundings. It is also found as a sequel to traumatism of various kinds resulting in suppuration and in general pyæmia, and it may follow direct traumatic injury to the brain. Cerebral abscess is usually single, except when it is produced by pyæmia. Although the abscess may occur in any part of the brain, a very common locality is in the cerebellum.

**SYMPTOMS.**—A cerebral abscess may exist for a considerable time without producing any symptoms which can be recognized during life. In cases where suppurative disease of the ear exists, a cerebral abscess may be suspected where, in addition to the temperature, which would naturally be raised from this process, the child's general condition becomes worse without any apparent cause, and where indefinite symptoms, such as mental dulness and irritability, arise. The temperature may also suggest the presence of imprisoned pus, and the probability of cerebral disease, in cases where the pus cannot be found elsewhere. Cerebral abscess may, however, exist for a considerable period without rise of temperature, and even with a subnormal temperature. It is apt to be slow in its progress and to cause general constitutional rather than local symptoms. Local symptoms produced by the presence of cerebral abscess are rare. When present, however, they are represented by headache, vertigo, mental dulness, vomiting, and convulsions, and are followed later by coma. When the abscess bursts into the ventricles, symptoms of sudden collapse appear, and death rapidly follows. Tremor and convulsions may occur in cases of cerebral abscess, but neither of them should be considered as in any way symptomatic of this condition.

**PROGNOSIS.**—The prognosis is very unfavorable unless the disease can be reached surgically.

**TREATMENT.**—The treatment should be operative if the abscess can be localized.

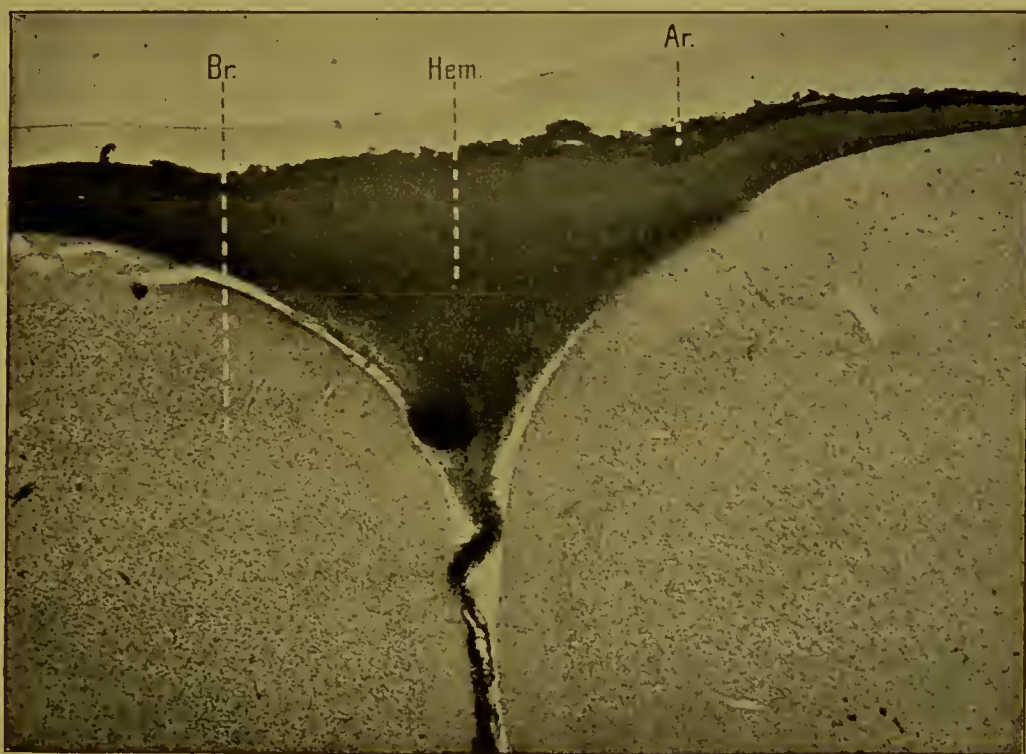
**CEREBRAL PARALYSIS.**—In using the term cerebral paralysis it must be understood that it is not intended to describe every disease of intra-cranial origin from which a paralysis may result. We may have a resulting paralysis from many intra-cranial lesions, such as hydrocephalus,



cerebral abscess, cerebral tumors, and other causes. The class of cases which I am about to describe under cerebral paralysis occurs usually in children under three or four years of age. In a certain proportion of these cases hemorrhage, embolism, and thrombosis are the causes of the acute symptoms. Most of the autopsies which have been made have shown sclerosis, atrophy, or porencephalia, which are probably secondary rather than primary. In these latter cases the original primary cause is not known. Cerebral paralysis results in a spastic paralysis involving one or more extremities, and may be in its distribution monoplegic, hemiplegic, paraplegic, or diplegic.

ETIOLOGY AND PATHOLOGY.—I shall first speak of the three known primary causes which I have just mentioned,—namely, (1) hemorrhage (rupture of one or more blood-vessels), (2) embolism (a foreign body brought to the brain from some distant part of the circulatory apparatus), and (3) thrombosis (an occlusion of one or more of the cerebral blood-vessels by a local coagulation of the blood).

FIG. 95.



Br., Brain. Hem., Hemorrhage. Ar., Arachnoid.

Of these three known primary causes hemorrhage is the most common. This hemorrhage is more apt to be meningeal than cerebral. It is for this anatomical reason that these cases of hemorrhage in infants are less liable to prove fatal than those which occur in adults. I have already described to you a case (Case 186, page 449) of presumably meningeal hemorrhage in an infant three days old. This case illustrated the possibility of recovery in even severe cases of intra-cranial hemorrhage.

Through the kindness of Professor Northrup I am enabled to show you this specimen (Fig. 95) of a case of meningeal hemorrhage in an infant born prematurely at the eighth month.

The mother had puerperal convulsions, and the delivery was by forceps. You see that the hemorrhage is in the locality where it is usually found in such cases, the *subarachnoid space* (vide Diagram 8, page 594). Intra-cranial hemorrhage in the infant and the young child may arise from various causes, such as increase of the intra-cranial pressure from various diseases, which produce stasis of the blood-current, or from traumata, whether from pressure or from direct injury to the skull and the brain. In addition to these causes, certain changes in the blood-vessels themselves, representing an atheromatous condition, are supposed to give rise to intra-cranial hemorrhage. Certain forms of degeneration may cause such a disorganization of the walls of the cerebral blood-vessels as to result in hemorrhage. In this latter class syphilis is a factor which must be considered, as must also, according to Sachs, general tuberculosis, meningitis, and cerebral tumors.

Next to hemorrhage, embolism is the most common cause of this class of cerebral paralysis. So few cases, however, have thus far been satisfactorily investigated by post-mortem examinations that I shall not dwell upon this condition, nor upon the still more rare resulting pathological lesion, thrombosis, except to explain that the emboli and thrombi act by cutting off the blood-supply of a certain portion of the brain, thus producing the disintegration of the cerebral tissue and the resulting paralysis.

As I have used the terms sclerosis and porencephalia, it may be well to define them.

*Sclerosis* consists of a shrinking and hardening of the cerebral tissues usually more or less strictly localized.

*Porencephalia* denotes a pathological hollow or depression in the brain running from the cortex towards the centre and usually communicating with a lateral ventricle.

The general pathological conditions to be remembered in cerebral paralysis, no matter what the original lesion, as has been so clearly summarized by Lovett in his paper on "Cerebral Paralysis in Children," are, *first*, a lesion of the brain involving, as a rule, some portion of the motor tract; and, *second*, atrophy and retarded development of the brain, with a descending degeneration of the lateral columns of the cord and pyramidal tracts. Finally, there is a possibility that the cause may be a defective development of the nervous centres.

Cerebral paralysis may occur in connection with a number of diseases, such as the acute exanthemata, pertussis, diphtheria, parotiditis, typhoid fever, and after continued convulsions. Difficult parturition, with or without the use of forceps, seems to be responsible for a certain number of the spastic cases, both paraplegic and hemiplegic.

**SYMPTOMS.**—Having made this preliminary explanation of the kind and extent of the knowledge which I am endeavoring to convey to you concerning the cerebral paralyses of infants and young children, I can now state the important general features of the disease which I should like to have you remember.



If the lesion has been of intra-uterine origin, we may get only the later manifestations of this lesion, just as we do in congenital syphilis. In like manner, if the lesion has occurred at the time of delivery, the primary symptoms are often masked, and the resulting symptoms of the more advanced pathological condition are noticed later.

Where the disease develops in extra-uterine life it is usually acute in its character and is marked by more or less fever, convulsions, and stupor. These early symptoms are merely those of a general nervous explosion following an irritation of the nervous motor centres. They may be the first manifestations of a disease of any kind, or they may occur in the course of one of the diseases of which I have spoken under etiology. If they happen to occur at night and are of short duration, they may be entirely overlooked, and the later symptoms of a cerebral lesion may be the first ones to manifest themselves. The child may die from the severity of these initial lesions before the later symptoms of paralysis have developed by which we can diagnose the disease. Screaming, vomiting, and delirium may at times usher in the attack. In the midst of or closely following these primary symptoms come the pronounced indications of a central nervous lesion, represented by hemiplegia (paralysis of an arm and a leg on the same side), paraplegia (paralysis of both legs), or diplegia (paralysis of both arms and both legs), cases of hemiplegia being the most common. In rare cases we find only one extremity affected (monoplegia).

In addition to the paralysis of the limbs, facial paralysis may occur either in hemiplegia or in diplegia, and, as a rule, spares the upper part of the face, so that the eyes can be closed and the brows raised, thus showing that it is not a peripheral facial paralysis. This form of facial paralysis often disappears early.

On examining the paralyzed limb we find a resistance to motion, the deep reflexes are exaggerated, and in most cases there is a feeling of rigidity on the paralyzed side. A few cases of flaccid paralysis have been reported. Sensation, as a rule, is not affected. When the child has come out of its stupor and the convulsions have ceased, it may be found to be aphasic. The intelligence is usually impaired, but this, of course, depends upon the location and extent of the lesion and the period when it occurred.

The intra-uterine and early infantile cases show the greatest mental disturbance. These children are apt to be very irritable, and, where the lesion is cortical, epileptiform convulsions are quite common. The electrical reaction of the muscles is normal. In the more advanced stages of cerebral paralysis additional symptoms begin to appear. The child learns to walk late, or, if it has already walked, the gait becomes peculiar. Rigidity followed by contracture of the flexor and adductor muscles may occur. In certain cases the spastic condition is so pronounced that the patellar tendon reflex and the ankle-clonus cannot be obtained. When walking is attempted, the patient is apt to stand on the toes, the knees knock together, and the spastic rigidity of the muscles produces what is called the spastic gait,



represented in its exaggerated form by the cross-legged progression, which is largely caused by the rigidity of the adductors of the thigh, and illustrations of which I shall presently show and explain to you.

The term spastic gait is applied to the peculiar way in which these children walk. In the more severe cases, when the child is placed upon his feet the contraction of the flexor muscles is excited to such a degree that he is unable to touch his heels to the ground, and stands on the ball of the foot and the toes, with his knees bent. This results in a clinging labored walk, in which the child's toes scrape along the ground and the feet tend to knock against each other on account of the contraction of the adductor muscles.

In the milder cases the same manner of progression occurs, but is more sudden and jerky, and the foot can be raised from the ground. Much unsteadiness thus results in these cases.

The affected limbs are apt to show some disturbance of their circulation, and some coldness. There are more or less atrophy and shortening of the bone, but to a less degree than in cases of poliomyelitis anterior. In a certain number of cases involuntary incoördinate movements are excited in the paralyzed limbs on voluntary effort (hemiataxia, Osler), and are usually designated as post-hemiplegic chorea. There may also be continuous movements (athetosis) of either a partial or a general variety. The sphincters are not affected, whether the case is one of hemiplegia or of paraplegia. The epileptiform convulsions which I have already referred to may appear quite early in cases of cerebral paralysis, but may also be delayed for a number of years, so that the possibility of these children becoming epileptic must always be considered.

DIAGNOSIS.—The general diagnosis of cerebral paralysis without regard to the special cause is of great practical importance to the practising physician, and should be thoroughly mastered before he attempts to diagnosticate the exact nervous lesion or to locate it with the precision of the skilled neurologist.

The diagnosis in a marked case of the disease is not difficult, but the determination of the exact lesion is often impossible after the period of onset has passed and we are left with a resulting paralysis. If facial paralysis is present, we can, as a rule, say that the lesion is in the brain; but this rule does not always hold good, as there have been very rare cases where this paralysis was present when the lesion was in the cord.

The symptoms on which we chiefly rely in making our diagnosis of cerebral paralysis are (1) the distribution of the paralysis, hemiplegic usually or paraplegic; (2) increased tendon reflex; (3) wasting comparatively slight; (4) normal electrical reaction; and (5) mental impairment.

The principal disease from which cerebral paralysis is to be distinguished is poliomyelitis anterior, and I shall in a later lecture when speaking of that disease explain to you the symptoms by which we can make a differential diagnosis between the two diseases by means of a table (Table 104, page 679). For the purpose of clearness, however, I will also state here that, in

contradistinction to the chief diagnostic symptoms of cerebral paralysis which I have just given you, you will find in poliomyelitis anterior (1) that the distribution of the paralysis is usually monoplegic; (2) that there is an absence of tendon reflex; (3) that there is an absence of rigidity in the early stages; (4) that there is rapid and marked wasting of the affected limbs; (5) that the reaction of degeneration is present; and (6) that there is no mental impairment.

In certain cases also a difficulty may arise in correctly understanding the relationship between cerebral paralysis and idiocy. The cerebral lesion is in many cases probably the same, but, according to its extent and location, we may have either (1) a cerebral paralysis alone; or (2) a cerebral paralysis accompanied by mental impairment or idiocy; or (3) idiocy without cerebral paralysis. There is a certain class of low-grade idiots in which some impairment of motion exists, apparently due to a mental inability to coördinate the muscles of the limbs properly. This may sometimes be accompanied by a diminution of sensation, which seems to be due to a want of perception in the higher nervous centres rather than to any actual lesion of the sensory tract. When the idiot's attention can be kept centred on the limb, the actual sensation does not seem to be much impaired. The differential diagnosis of this condition occurring in idiots from cerebral paralysis is easily made, for it exists in those cases only of the former where the mental development is much impaired, and it is not, as a rule, accompanied by true paralysis, as there is no weakness, but simply incoördination; in these cases also the tendon reflexes are, as a rule, not increased.

Cerebral paralysis can be diagnosticated from the paralysis which occurs in connection with caries of the spine, principally by the presence of cerebral symptoms in one case and the prominence of the spinal vertebræ and the rigidity of the spine in the other.

I should also mention here that the rare cases of syringomyelia may be mistaken for cerebral paralysis. The points of differential diagnosis in these cases are that in syringomyelia, although the weakness of the limbs may be so extensive as closely to simulate paralysis, yet the diminution of thermic sensation, which I shall presently speak of when describing the disease to you (page 690), easily distinguishes it from the normal sensation which is present in cerebral paralysis in cases where the test for sensation can be employed. The disease, however, is so rare in children that it need not be dwelt upon.

PROGNOSIS.—The question which immediately arises when the physician is confronted with a case of paralysis in an infant or a child is, What will be the result of this attack? not, What is the special anatomical lesion which is causing it? Knowing, as I shall presently explain to you, that where the lesion is of spinal origin the chances for recovery are fairly good, you will at once appreciate the vast difference which your answer may make to those interested in the child when you state that the disease is in the cord and that recovery is probable up to a certain point without mental impairment,



rather than that the brain is involved and possibly mental as well as physical incapacity for life may result. The prognosis for life in cerebral paralysis is soon determined in the early days of the attack, and of course depends on the location and extent of the cerebral lesion. Entire recovery is rare. The leg, as a rule, recovers more rapidly and to a greater extent than the arm, which seldom regains its entire usefulness. The spastic rigidity usually goes on to decided contracture. In some cases no mental change is apparent; in others the mental development is merely retarded, and the child learns to talk some years later than is normal. In a large number of cases, however, the mind is much enfeebled. The occurrence of epilepsy as a result of cerebral paralysis is so common that it should be especially mentioned in this connection, as it makes the prognosis much more serious both as to the degree to which the mental impairment may attain and as to the life of the patient.

Except in very rare cases, the children can be taught eventually to walk.

**TREATMENT.**—The treatment of cerebral paralysis must necessarily be unsatisfactory. It is to be directed to keeping the paralyzed limbs in as good a condition as possible and thus avoiding contractures of high grade. This can be accomplished in a measure by patient and continued massage and manipulation, chiefly in the direction of stretching the flexor muscles and cultivating the use of the extensors. The faradic current used three or four times a week for five or ten minutes is a useful adjuvant, and, if necessary, surgical interference to relieve undue tension of the flexor tendons is indicated.

The mental training of these cases is exceedingly important, and should be attended to carefully. In this connection it is well to remember that the division of the contracted tendons in some way seems to improve the mental condition. Trephining the skull over the supposed seat of the lesion does not, with our present knowledge of the usual nature of these lesions, present a particularly encouraging outlook. The few cases which have been operated upon have not been benefited.

It should be thoroughly understood that surgical operations to relieve the contractures do not influence favorably any pre-existing paralysis or incoordination, but that it often puts the limbs in a condition in which massage and electricity can be applied to greater advantage. The indications for the division of the tendons of the contracted muscles exist when the contracture is so firm that thorough treatment by massage and electricity produces no essential relief. Cutting the tendons in cases of low grades of idiocy has usually been considered contra-indicated, but this opinion is not shared by all observers, as in a certain number of cases at the Boston Children's Hospital decided benefit has been found to result.

I have thus endeavored to give you a precise and practical idea of a very complicated subject. In order to do this I have used as few names as possible and have avoided many plausible but theoretical explanations of noted writers. As an instance of this, I have passed over Strümpell's brilliant



but unproved theory of *polioencephalitis* as one of the causes of cerebral paralysis. You will, however, now understand how inadequate are the various names, such as *spastic paralysis*, *spastic rigidity*, *spastic diplegia*, *Little's disease*, and *infantile hemiplegia*, to cover the broad range of pathology and symptoms which is represented by the class of cerebral cases which I have designated under the general term *cerebral paralysis*.

I have a number of cases here to show you which represent this condition of cerebral paralysis in children.

The first case is a boy (Case 294), five years old. Up to the age of seven months he is said to have been in a normal condition. The disease which was followed by the symptoms which he now presents occurred when he was seven months old. At this time he was attacked with fever and a convulsion, and later was found to have paralysis of the right arm and both legs.

CASE 294.



Cerebral paralysis. Right hemiplegia, with affection of opposite leg. Male, 5 years old.

On examining the child you see that the thumb is turned in on the palm of the hand and the fingers are slightly flexed and at times slightly extended. Both legs are somewhat flexed at the knees. The hamstring tendons are tense and unyielding. The knee-jerks are increased. The heels are raised from the ground.

This child is a case of hemiplegia with affection of the opposite leg.

In mild cases of this kind the treatment is by massage and electricity; in the more severe forms apparatus is required. In a very severe form like this, operative interference

is necessary before massage, electricity, or apparatus can be applied with advantage. In this case division of the hamstring tendons and of the Achilles tendons is indicated. Operative proceedings in cases of this kind must be recognized as only rectifying the position and preparing the limbs for further treatment by massage, electricity, and apparatus.

The next case (Case 295) is a girl, five years old.

She has a good family history. The labor was easy, and was not instrumental. She developed well and was healthy until she was ten months old, when it was noticed that she did not move her arms as she ought to, that she did not use her left arm at all, and that the left leg was not used as well as the right. This condition has persisted.

CASE 295.



Cerebral paralysis. Diplegia. The left extremities affected more than the right. Female, 5 years old.

On examination you see that she has strabismus. She cannot hold her head up straight. She cannot sit up alone or stand. Her head is small and narrow, and has a long antero-posterior diameter. The reflexes are increased. The power of her left arm is much impaired, and there is some contraction of the fingers and elbow of a spastic character. She does not move her left leg well. The sensation is dulled alike in both legs. Her face has an idiotic expression, she is poorly developed mentally, and she cannot talk.

She shows the form of spastic cerebral paralysis which is called diplegia, the left extremities being more affected than the right. The face is not involved in this case.

The prognosis of a case like this is unfavorable so far as recovery is concerned, on account of the great mental impairment. Operative treatment is, however, indicated, as at times improvement results in even decidedly idiotic cases.

This little girl (Case 296), two years old, was born after a severe instrumental labor.

She has always from birth shown weakness of the arms and legs. She was unable to sit up until she was a year old, and she has never stood or walked. Her intelligence is apparently normal. You see that the cranium is normal in shape, that the parietal eminences are somewhat enlarged, and that the fontanelle is still open. There is no disturbance of the facial muscles. The right leg is slightly larger than the left. The teeth are in excellent condition. The upper extremities appear alike, but she cannot loosen her fingers after grasping an object with her right hand. The epiphyses of the wrist are much enlarged, and those at the ankles are slightly so. There is no definite rosary. The back is

CASE 296.



Cerebral paralysis. Congenital cerebral diplegia and rhachitis. Female, 2 years old.

somewhat rigid. There is no marked deformity. There is a tendency to rigidity in both lower extremities. The feet are inverted. The patellar reflexes are increased. Sensation is normal. She can use her hands well, except as above described.

She represents the class of cerebral paralysis which is called cerebral diplegia. You see that she is also rhachitic.

Here is a little boy (Case 297, page 658), four years old, who was perfectly well at birth, but who when he was six months old had a number of convulsive attacks without any known cause.

When he was two years old he had an attack of measles, followed by varicella, and later by pertussis. He has never been able to sit or stand alone. He is fairly developed and nourished, and his intelligence is normal. He has marked general kyphosis when supported by the arms. When he is assisted to walk he also shows the condition of cross-legged progression. The arms are somewhat stiff, and he holds the forearms slightly pronated. The triceps reflex is somewhat increased. The legs are usually held somewhat flexed on the body, and the knees are also slightly flexed, with the feet in the position of slight equinus. The knees are held closely together. Rigidity is less marked in the right



leg than in the left. The patellar reflexes are much increased, and ankle-clonus is present. There is very marked rigidity of the left side, so that the reflexes are obtained with difficulty.

The treatment in such cases as this, where there is no mental impairment, should be operative. Section of the adductors of the thigh, of the flexor tendons of the knee, and of the Achilles tendons is indicated.

This next boy (Case 298), five and one-half years old, has nothing in his family history that bears upon the disease with which he is affected.

Nothing of an abnormal nature was noticed about him until he was fifteen months old,

CASE 297.



Cerebral paralysis. Diplegia. Cross-legged progression. Male, 4 years old.

CASE 298.



Cerebral paralysis. Spastic paraplegia. Cross-legged progression. Male, 5½ years old.

when it was found that he could not walk. He had more or less mental impairment, nystagmus, stiffness of the adductor and flexor muscles, and paresis of the extensors of the lower extremities. The knee-jerks are much increased, and there is slight ankle-clonus. He walks in the characteristic manner called cross-legged progression.

When an infant he evidently had some cerebral lesion, and he represents very well what I have explained to you as spastic paraplegia. There will probably never be any improvement in his physical condition, and his mental state will always be unsatisfactory.

This boy (Case 299), six years old, has no history of any hereditary disease.

He was healthy at birth, but the labor was a severe one, and was terminated with instruments. He developed normally during the first two years of his life, and walked when

he was eighteen months old. He is stated to have had convulsions in his third year, and these convulsions occurred again when he was four years old. They were followed by the paralysis for which he has come to the hospital to be treated. He does not use his left hand well, and the grasp of the left hand is less strong than that of the right. The triceps reflex is exaggerated on both sides. The left foot can with difficulty be flexed dorsally. The right knee-jerk is normal, the left is increased. He has flat-foot, and walks with his left foot rotated in. He is now six years old, and is otherwise well and strong.

This is a case of left spastic hemiplegia.

The treatment in this case is by massage and electricity. Apparatus does not seem to be indicated, as its only use is to support the limbs or to correct deformity.

This boy (Case 300) is four years old. There is a history of phthisis on the maternal side.

CASE 299.



Cerebral paralysis. Left spastic hemiplegia, two years' duration. Male, 6 years old.

CASE 300.



Cerebral paralysis. Spastic paraplegia. Male, 4 years old.

His mother has four other, healthy children, but has a history of three miscarriages. This child was born prematurely, and the delivery was instrumental. He has always been delicate, and had an attack of measles one year ago. He did not attempt to walk until he was three years old, and it was then noticed that he did not use his legs well. He is mentally normal. His arms appear to be normal. When placed on the floor he gets up in a manner like that which is shown in cases of pseudo-hypertrophic muscular paralysis. When he stands his knees are highly flexed and adducted. He walks on his toes, with a tendency to cross the knees. This tendency can be only partially overcome. There is no



apparent atrophy of the muscles. The knee-jerks are slightly increased, and there is slight ankle-clonus. The skin shows some disturbance of circulation.

He represents the class of cerebral paralysis which has been designated spastic paraplegia, the original cerebral lesion having affected the legs only.

If this child's condition is not much improved by passive movements of the limbs and massage, it may be advisable to resort to operative treatment and divide the tendons of the flexor muscles.

I happen to have here in the wards a case which apparently represents the symptoms of traumatic hemorrhage.

This little girl (Case 301) is four years and nine months old. She was brought to the hospital February 28, with a history of having fallen from the roof of a three-story building upon a brick sidewalk. She was unconscious. She vomited slightly, and she was found to have an ecchymosis on the left side of her head. Her pupils were equal and reacted to light. Her respirations were rapid; the extremities were cold. She moved all her limbs vigorously. Some clotted blood was found in and about the nostrils. The temperature was 36.3° C. (97.4° F.); the pulse was 90, and the respirations were 26. She ground her teeth and cried out in the night. The muscles of the left arm and leg moved actively.

On the next day it was found that she could swallow milk. She passed her urine involuntarily. She was still unconscious, and the movements of the left arm and leg continued.

On the following day, for a short time the right pupil was larger than the left, and would not react to light. Although she could not speak, her eyes would follow the finger; the eyes also had a restless movement. An enema produced a passage of a small amount of feces and a few drops of blood. The respirations were very deep, and the face was flushed.

On the following day she still continued to move her left arm and leg, while the right arm and leg remained passive. The pulse was irregular and intermittent. She was reported to have slept more than at any time since the accident. She was still unconscious, but was less restless.

On the next day the pulse was irregular, as it was also two days later. The pupils were irregular, and she opened her eyes and fixed them on objects at times. She also rolled her eyes and yawned. She was still unconscious.

Two days later she had slight opisthotonos, and there were spasmodic movements of the left arm and leg. She slept a great deal.

On the following day she appeared brighter, and followed objects with her eyes. Her pulse was irregular, from 80 to 90.

Two days later she seemed brighter, and moved the left arm and leg less. She also made voluntary movements, such as to push objects away from her. On this day she gave evidence that she understood what was said to her. Three days later she seemed to recognize her mother.

On the following day she began to use her right arm very slightly. She ate a cracker, and was at times quite conscious.

The next day she appeared more intelligent, and on the day after that she began to speak single words. It was found, however, that she could move her right arm but very slightly. Since this time she has always been perfectly conscious, endeavors to say words, and notices the children in the wards, as well as her playthings.

To-day,—the twenty-ninth day from the time when the accident occurred,—as you see, she can walk, though with difficulty, as the right leg is very unsteady.

She apparently has had a lesion on the left side of the brain, represented by a hemorrhage and caused by traumatism.

(Subsequent history.) One week later she was discharged from the hospital. At that time she could use the right arm fairly well, but walked with some difficulty on account of the weakness of the right leg. Her articulation was labored, and her pupils were unequal.



**ATHETOSIS.**—Athetosis is a symptom, and not a disease, and is represented by continuous incoördinate arrhythmical movements of the extremities, the face, and the body. This condition may be acquired or congenital. The acquired form may follow cases of hemiplegia or diplegia, in which event it affects the paralyzed limbs. Certain cases of acquired athetosis occur without any accompanying paralysis. In congenital athetosis, and in the acquired form without paralysis, the symptoms usually begin in the first year.

**PATHOLOGY.**—The pathological condition which exists in cases of athetosis is supposed to be a chronic cerebral irritation in the neighborhood of the basal ganglia and in the internal capsule. The condition as we see it clinically, therefore, is wholly a symptom of some organic lesion of the brain.

**DIAGNOSIS.**—The diagnosis of *acquired athetosis* is made by the character of the movements. These are continuous, and are distinguished from those of chorea by being vermicular and less spasmodic.

The diagnosis in cases of *congenital athetosis* is not difficult, as in no other disease does an infant present at birth these peculiar movements and this grotesque form of flexion and extension of the fingers and toes. The disease called *congenital chorea*, in which involuntary arrhythmical movements exist, is distinguished from athetosis by the character of the movements, which resemble those of ordinary chorea.

CASE 302.



Congenital athetosis. Female, 2 years old.

**PROGNOSIS.**—The prognosis of athetosis in regard to recovery is unfavorable. So far as the general health is concerned, the individual may develop fairly well and may live for years, as in the case of a man, twenty-two years old, reported by Bullard.

**TREATMENT.**—There is no known treatment which has proved to be of benefit in children. As they grow older the training of the affected limbs

may be undertaken, but, as a rule, the results are unsatisfactory. Massage and electricity have proved to be of no value.

I have here a little girl (Case 302, page 661) who represents this condition of congenital athetosis.

She is two years old. She has never had any acute disease. She was born after a normal labor, and has received no subsequent injury. She has never talked nor shown much interest in her surroundings, nor has she been able to sit up or hold up her head without support. The bowels have always been regular and the appetite good. She is well developed, and, as you see, well nourished.

The disease is characterized by the continual incoördinate arhythmical movements of the head, trunk, and extremities; these movements are often quite rapid. There is constant flexion and extension of the hands and fingers, the fingers at times being bent backward and assuming most grotesque positions. This phenomenon is also seen in the toes. The expression of the face, as you see, is not that of ordinary intelligence. I find that I cannot determine the reflexes, on account of the resistance of the child to examination. She is usually irritable, but occasionally smiles slightly and takes some slight notice of those who are near her.

The prognosis in this case, so far as recovery is concerned, is bad. There seems to be no especial reason why she should not live.

**INTRA-CRANIAL TUMORS.**—In infancy and early childhood tumors of many varieties may occur in the brain and its meninges. The most common form of intra-cranial tumor is tubercular. The next in frequency are gliomata, sarcomata, and glio-sarcomata. The other varieties, such as eareinoma, lipoma, myxoma, and teratoma, are very rare; and syphilitic gummata, which are so frequent in adults, are exceedingly rare in infancy and early childhood. The parasitic cysts in the brain which occur quite frequently in individuals in other parts of the world, especially in Germany, are seldom met with in this country.

These tumors may be either of intra- or extra-uterine origin. Of these the tubercular is the most common.

**PATHOLOGY.**—The *tubercular* tumors of the brain or its meninges are, as a rule, secondary to a tubercular growth in some other part of the body, or to tubercular disease of some part of the skull, such as the orbit or ear. These tubercular tumors may be single or multiple, the latter being the more common variety. They may be found in any part of the brain or its meninges, and occur with especial frequency in the cerebellum. They may vary in size from a small collection of miliary tubercles to much larger masses. When one or more cheesy masses of a tubercular nature are found in different parts of the brain, the condition is called solitary tubercle. The *gliomata* grow most frequently in the white substance of the brain, but sometimes develop in the gray matter. According to Starr, they grow less rapidly than sarcomata, and never involve the membranes. They are usually primary, but may develop as secondary to glioma of the retina (Starr). The sarcomata are both of the round-celled and of the spindle-celled variety. Although not quite so frequently found as the gliomata, they are more frequent than the glio-sarcomata or myxomata. They are usually



round in shape, and develop both in the nervous tissue and in the cerebral membranes, and in both the white and the gray matter of the cerebrum and cerebellum. The other varieties of tumor of the brain are so rare that they need not be considered here.

In connection with intra-cranial tumors, I might mention that *intra-cranial aneurisms*, according to Starr, are rare in childhood and are never very large. They increase in size rather more rapidly than aneurisms elsewhere, and show a tendency to rupture. They are found upon the larger arteries of the base of the brain and on the Sylvian arteries. The pathological condition of the brain in the neighborhood of these growths is such as would result from the impediment to the blood-current in the small vessels, or from compression of some of the larger arterial trunks. The condition is usually one of anæmia. The anæmia may be sufficient to impair the nutrition of the nervous tissue. As a still later pathological condition in these cases produced by pressure, areas of atrophy of the brain may occur.

**SYMPTOMS.**—The symptoms which result from intra-cranial tumors are very numerous, and are rendered all the more difficult to recognize in infancy and early childhood by the pronounced nervous phenomena which may result from even a slight degree of irritation or pressure in the young and undeveloped brain-tissue.

The general symptoms vary very much in accordance with the size and vascularity of the tumor, and according as it is growing or has become stationary. In the former case the symptoms are often apt to be more severe than later, when, the tumor having become stationary, the brain-tissue adapts itself to the new conditions produced by the morbid growth. Intra-cranial tumors in infants and in young children are often latent, present no symptoms, and are sometimes discovered only after death. A certain number of cases, on the other hand, present only general symptoms, such as headache, cerebral vomiting, attacks of vertigo, convulsions, and optic neuritis, which cause us to suspect intra-cranial disease, but give an indefinite idea of its location. Again, these tumors may produce local symptoms in addition to the general ones. These local symptoms are represented by paralyses of different kinds, anomalies of sensation, affections of the special senses, and staggering. These later symptoms arise according to the site of the tumor and the parts of the brain which are affected by it, and by means of them we can more or less approximately judge of its situation, size, and rapidity of growth.

I shall not enter here into the various complex questions of brain localization, but shall refer you for further information to works especially devoted to that subject (Keating's "Cyclopædia of the Diseases of Children;" Starr). I may, however, say that paralyses of the extremities are caused by an affection of the motor cortex, the internal capsule, or any portion of the motor tract on the opposite side of the brain above the crossing of the pyramids. Staggering or cerebellar ataxia is suggestive of cerebellar disease, while the



involvement of the intra-cranial nerves suggests a tumor of the base of the brain or pressure on these nerves at some point, and more rarely an affection of their nuclei. The tendon reflexes are apt to be exaggerated, but in some cases are normal, and in others are said to be absent. The symptoms of cerebellar ataxia which at times occur where the tumor is situated in the cerebellum consist of a staggering gait resembling that of an intoxicated person, the steps being irregular in length and the body swinging from side to side. The child in these cases has a subjective sense of falling or turning back, and grasps for support or sinks into a chair or to the floor. This form of ataxia is to be distinguished from that which is found in spinal disease, and which is due to an inability to coördinate the muscles of the lower extremities properly. This latter form of ataxia is much more regular than the former, each step being insecure and unsteady, but without the violent and sudden reeling, after two or three steady steps, which occurs in the cerebellar form.

In young infants a tumor may cause a protuberance of some part of the skull by pushing one of the bones outward, as was seen in a case (Case 303), eight months old, of teratoma which was operated upon by Dr. Lovett at the City Hospital, and which is one of the few instances of this form of tumor on record.

DIAGNOSIS.—The diagnosis of tumors of the brain must in the great majority of cases be made by elimination. The variety of tumor can be determined most readily by considering the history of the case, as to whether it is tubercular, syphilitic, or otherwise. The diagnosis of a tumor can often be made by the slow and gradual development of the disease. When severe headache and vomiting exist, followed by paralysis, either monoplegic or hemiplegic, especially if this paralysis develops slowly, we should suspect the presence of some form of intra-cranial growth. This suspicion is much strengthened by the presence of optic neuritis or optic atrophy. The presence of localized convulsions in such cases tends to confirm the diagnosis, while if marked ataxia exists we are justified in suspecting cerebellar disease. A normal or only slightly elevated temperature with these symptoms which I have just mentioned also points to the diagnosis of a cerebral tumor.

PROGNOSIS.—The prognosis of tumors in early life is very unfavorable, no matter what the variety of the tumor may be. Although the patient may for a long time remain wholly unaffected by the morbid growth, he eventually, except in rare cases, succumbs to the disease.

TREATMENT.—Surgical interference in children, as in adults, proves on the whole to be the most valuable means at our command for lengthening life in cases of cerebral tumors. There is no other treatment which is of any especial benefit in either retarding the growth or curing this class of cases. Even where the exceedingly rare form of syphilitic gumma exists, iodide of potassium and other drugs have not apparently proved to be of much value.

In regard to what I have said concerning the latency of tumors of the brain, the case which I showed you in the wards some days ago exemplifies the extent to which this latency can exist where the tumor is tubercular.

I have to-day the opportunity of presenting to your inspection the results of the autopsy on this case.

You may remember my telling you when I was examining this infant (Case 304) while alive that I could detect nothing abnormal except a moderately raised temperature by which I could distinguish it from the case of infantile atrophy in the next bed, which had an almost identical temperature and similar symptoms.

The infant was thirteen months old, had never had any especial disease, and entered the hospital weak and emaciated. Its mind was clear. Its pulse was weak but regular, and neither slow nor quick for its age. Its temperature was at times somewhat raised, varying from 37.2°–38.4° C. (99°–101° F.). There were no convulsions, and no paralysis or contractures, but merely progressive loss in weight, and finally death.

An examination of the brain of this infant shows miliary tubercle of the pia mater at the base of the brain without acute inflammation, which accounts for the lack of acute cerebral symptoms. Of especial interest, however, in the case are the patches of solitary tubercle, 1.2 cm. ( $\frac{1}{2}$  inch) in diameter, which you see in the left temporal and occipital lobes and in the right frontal lobe of the cerebrum, and also in the lower left cerebellum. There is also caseous tubercle of the post-bronchial glands, tubercle of the lungs with a slight amount of broncho-pneumonia, miliary tubercle of the pleura, liver, and spleen, and caseous tubercle of the mesenteric glands.

Through the kindness of Dr. Bullard I am enabled to show you the result of the post-mortem examinations in some cases of cerebral tumors which have just occurred in his practice.

A boy (Case 305), four years old, of healthy parentage, but with a history of tuberculosis in his grandmother and an aunt, was perfectly well until he was ten months old. At that time he had an attack of general tonic convulsions followed by paralysis of the right lower leg. After that the right leg slowly improved, but never entirely recovered. He began to walk when he was fourteen months old. After this first attack he remained perfectly well until two months before his death, when he was found to have ptosis and acute conjunctivitis on the left side. Three weeks before his death he began to lose in weight and to be very sleepy and stupid; he was feverish and lost the power of walking; he also lost his appetite and his bowels were very constipated. There was no history of his ever having had any disease of the ears.

When examined by Dr. Bullard the head was not retracted, and no tenderness was found anywhere over the cranium. When the left eyelid was raised the eye was found to be turned upward and outward. There was some swelling of the eyelids. Both eyes reacted to light. The tongue was protruded straight. The heart and lungs were normal. Nothing abnormal was found in the abdomen or spine. There was a flaccid paralysis of the right lower extremity, with foot-drop. Nothing abnormal was found in the urine. A few days later there was found to be some loss of power in the left upper extremity and left toe-drop. The knec-jerks were present.

He was treated with iodide of potassium, and his general condition improved somewhat. The drowsy condition, however, returned, and, although for a time improvement took place in regard to the movements of his limbs, he gradually became more stupid, and finally was in a torpid condition. He swallowed with great difficulty. He had strabismus of the left eye. Nothing abnormal was found in the urine, but it was passed, as well as the feces, involuntarily. His temperature varied from 37.2° to 37.7° C. (99° to 100° F.), and his pulse was between 80 and 90.

An examination during the latter part of his life showed that the thoracic, epigastric.



cremasteric, and plantar reflexes were excellent; the triceps reflexes were good. The knee-jerks were good, the right less than the left, the latter being exaggerated. Nothing else abnormal was detected.

An examination of the eyes by Dr. Standish, thirteen days before his death, showed marked choroiditis in the right eye, with large tortuous veins and arteries nearly obliterated. There was indistinctness of outline in the disk in the left eye, with the veins large in proportion to the size of the arteries. At this time he had deep sighing respirations with intervals of a minute or more.

One week before his death the right arm was rigid at the elbow and the hand and fingers were flaccid. At times an erythema would be seen on his arms and body. Turning his head evidently caused pain. The upper part of the head was cyanotic. He was much emaciated. The right pupil was much larger than the left, and neither pupil reacted to light. The pulse increased in frequency, and at times was between 158 and 160. The abdomen was retracted. He remained in a stupid state until his death.

The post-mortem examination made by Dr. Bullard showed rigor mortis present in a moderate degree. The abdomen was retracted. The head was larger than normal in proportion to the size of the body. Nothing else abnormal was noticed on physical examination.

The pleura and pericardium, with their cavities, and the heart were found to be perfectly normal. Behind and to the right of the trachea, at or just above its bifurcation, two nodules about 2.5 cm. (1 inch) in diameter were found; they were apparently enlarged lymph-glands. On section they were found to be composed of yellowish-white cheesy material. Nothing abnormal was detected in the right lung. In the left lung, about the centre of the upper lobe, was a cavity about 2.5 cm. (1 inch) in the longest and 1.2 cm. ( $\frac{1}{2}$  inch) in the shortest diameter. This was filled with cheesy material, friable, and easily removed. The liver, spleen, intestines, gall-bladder, and bladder presented nothing abnormal.

On examining the head there was nothing abnormal noticed externally. The longitudinal and lateral sinuses contained a very small amount of blood, clotted and liquid. The dura mater everywhere seemed normal, and was not unusually adherent to the cranium. The pia mater seemed normal everywhere except in the neighborhood of the Sylvian artery. Here it was more adherent than elsewhere, small pieces of the brain coming away with it when it was torn off. Both lateral ventricles were enlarged.

On the superior surface of the cerebellum there was a projection in the median line of part of a mass which occupied the anterior portion of the central lobe. On section it was found to be yellowish-green and much firmer than the rest of the cerebellum. Nothing else abnormal was detected macroscopically.

The tumor was examined by Dr. Dunham, who reported that it appeared to have occupied the anterior middle portions of the cerebellum, and to be about 5 cm. (2 inches) broad, 3.5 cm. ( $1\frac{3}{8}$  inches) from in front backward, and 2.5 cm. (1 inch) from above downward. It was circumscribed, and its substance was more consistent than that of the cerebellum. It had two globular projections 1.2 cm. ( $\frac{1}{2}$  inch) in diameter, one on each side, extending forward, probably one towards each side of the upper part of the fourth ventricle or beginning of the aqueduct of Sylvius, but not far enough to invade the pons. It did not extend farther back than the limits of the quadrate lobes. The cerebellar peduncles were not involved. The amygdalæ, which were almost directly below and in front of the tumor, were not affected.

Histologically the tumor was a sarcoma. In parts the structure was gliomatous; in others the cellular elements were so abundant that the microscopic picture was like that of a small, round-celled sarcoma. There were many blood-vessels in the substance of the tumor. Although the lung was not much affected, several of the peribronchial glands had undergone cheesy degeneration. The kidneys were small, but their tissues showed nothing unusual. Nothing abnormal was found in the other organs.

There was no evidence of tubercle in the brain or its meninges.

The next case is that of a little girl (Case 306), eleven years old, a patient at the Children's Hospital. Her parents were healthy, and there was no history of any disease affect-



ing the nervous system in the family on either side. There was no history of phthisis. The child was born after a natural labor, with a head presentation, and without the aid of forceps. When she was eighteen months old she had an attack of pneumonia: she is said to have had some "head trouble" at that time, and was never well afterwards. Up to the age of six years she had earache, accompanied with a discharge from the ear. According to Dr. Bullard, there was some evidence of hydrocephalus at or before this time. She was never as strong as other children. She did not walk until she was twenty-seven months old, and she was more liable to fall than other children. She was always of a nervous temperament, restless, and unable to sleep well. She could never bear any excitement. When she was seven years old she had another attack of pneumonia, with a complicating pertussis.

Three years ago she had a severe illness, of which the most prominent symptom was pain in the head. This pain was intense in the temples, especially in the left one, and she would hold the back of her head with both hands. There was much severe vomiting at this time. The temperature was stated to be about normal, and the pulse natural. There was also pain in the neck and in all the limbs, but it was slight in the right extremities and more severe in the left extremities. This illness lasted ten weeks, and she never completely recovered from it. She, however, became well enough to go to school.

A little later she was found to be blind in the left eye, and three weeks later the right eye also became blind. The blindness was supposed to have come on gradually.

When she was between eight and nine years old she had another very severe illness, characterized by pain in the head and vomiting. At this time she was first noticed to have momentary "spasms," in which she would scream with pain and would then lose consciousness, but without convulsions or rigidity. There was no heightening of the temperature during this illness. During this attack she could not move any of her limbs.

A few months later she began to improve, and a month after this was able to walk alone. After this there was gradual improvement.

When she was ten years old the headaches became worse, and she had a third severe attack, with vomiting and pain in the head, lasting four weeks. Since that time she has not been able to walk alone.

On entering the hospital she was found to be totally blind. There were paresis and incoördination of both lower extremities. There was considerable incoördination of the left hand, while coördination of the right hand seemed normal. There was no atrophy anywhere. The sensation was unimpaired. The knee-jerks were alike and normal. For two weeks she was unable to go to sleep easily, on account of pain and restlessness. While in the hospital she would have nausea and vomiting at times, and headache would occur four or five times a week, but not so severe as to make her scream. She was unable to walk without assistance. When some one held her hand she walked with the feet quite straight, striking the ground first with the heels, and tilting the pelvis more than normal. Her appetite was good. At times she would have constipation, followed by diarrhoea, with involuntary dejections. The vomiting and headache continued. While she was in the hospital she was for a time quite comfortable. Her temperature ranged from 36.9° to 37.7° C. (98.5° to 100° F.). There were no other symptoms worthy of note. Examination of the urine showed it to be normal.

After leaving the hospital, when she was eleven years old, she had less headache for a short time, but then became worse. She had several severe attacks, reported by the family as "fainting-spells," in which there was loss of consciousness without convulsions, and she died quietly in one of these to-day.

I have here the result of the examination of the head and a statement of the pathological conditions which were found.

On removal of the external tissues the cranium presented a translucent appearance, suggesting extreme thinness of the cranial bones, and large white bands 2.5 cm. to 3.7 cm. (1 to 1½ inches) broad lay in the position of the larger cranial sutures, as though these sutures had long been held open by intra-cranial pressure. The bones of the cranium were unusually thin, those forming the calvaria being not much more than 0.6 cm. (¼ inch) in thickness. The calvaria was very elastic, could be readily compressed, and when the pressure was

removed would spring back to its original shape with much force. The inner and outer tables were thin and very hard, while the diploë seemed disproportionately large. The dura mater was adherent along the coronal sutures, but was otherwise normally free. Its blood-vessels were rather injected. The longitudinal sinus was empty. The pia mater showed nothing abnormal, except that its blood-vessels were somewhat injected. A large quantity of clear, pale-yellowish fluid, estimated at about 1440 c. c. (3 pints), escaped from the cerebral ventricles on removal of the brain. The third ventricle was much dilated, and formed a cyst-like projection at the base of the cerebrum. The lateral ventricles were greatly dilated, each occupying almost the whole of the corresponding hemisphere, the white substance and the cortex between them being much thinned. There were no hemorrhages, cysts, or other peculiarities detected in the cerebrum.

On inspection of the cerebrum, a gelatinous mass of rounded lobular shape, suggesting a cyst, was seen projecting from the external surface of the left lobe. On palpation this was found to contain fluid, and to be connected with a hard mass occupying this lobe. This mass was examined by Dr. Mallory, who reports that the cyst which I have just mentioned was emptied and collapsed. On section vertically through the centre of the left lobe of the cerebellum, extreme resistance was met with, such as would suggest bone or cartilage. The section showed a globular cavity 3.7 cm. ( $1\frac{1}{2}$  inches) in diameter, containing a thick, greenish-yellow, semi-fluid mass, resembling pus, and surrounded by a circular border, 3.7 cm. ( $1\frac{1}{2}$  inches) broad, of a yellowish-white color with a slight bluish tinge, largely composed of circular masses like sago-grains, separated from each other by tissue of nearly the same color as themselves. These circular masses gave a peculiar translucent appearance to this border or capsule. The tumor occupied the larger portion of the left lobe of the cerebellum and its whole outer two-thirds.

The report of the microscopic examination made by Professor Councilman is as follows:

The tumor is not so sharply circumscribed as the macroscopic appearances would indicate. The structure of the tumor itself is somewhat complex. It consists of a variety of cells, the prevailing type being similar to those of round-celled sarcomata. This is especially seen in the portions of the tumor apparently the freshest and of most rapid growth. In some places the cells are rather irregular in size, with numerous processes similar to the spider-cells of the brain. The principal extension of the tumor is along the lymph-sheaths of the vessels. These are filled with round cells, in many places at a considerable distance from the main body of the tumor. There is more or less tissue between the cells, consisting in part of a regular formation of close connective tissue and in part of a very loose reticular tissue. Throughout the tumor there are numerous foci of degeneration, the largest of which correspond to the circular masses described by Dr. Mallory. In numerous places in the tumor there is an entire infiltration with pus-cells. One of the chief characteristics of the tumor is the hyaline degeneration both of the cells and of the blood-vessels. Large masses of a perfectly homogeneous material giving all the reactions of hyaline are found both in and along the course of the blood-vessels in various parts of the tumor. From the size and position of many of these hyaline masses it is evident that cells also have taken part in their formation.

The tumor is to be regarded as a glio-sarcoma, with hyaline degeneration of the blood-vessels, and foci of necrosis.

**INTRA-CRANIAL SYPHILIS.**—Intra-eraniel syphilis may be either congenital or acquired. According to Bullard, the intra-eraniel lesions are essentially the same in both forms.

**PATHOLOGY.**—Intra-cranial syphilis may be divided pathologically into three forms: (1) diffuse inflammation of the meninges or their neighboring tissues, (2) localized growths or tumors (gummata), and (3) syphilitic endarteritis. In the latter case (endarteritis) there may be local dilatation or local occlusion of the blood-vessels. These conditions are apt to occur simulta-



neously. When the dilatation reaches an advanced stage a thinning of the arterial walls results, which may lead to rupture of the blood-vessels or to hemorrhage. More common than the hemorrhage, however, is the occlusion of the blood-vessels, which cuts off the blood-supply and acts in the same way as in other cases of thrombosis of the arterics, causing more or less softening and disintegration of the cerebral tissues supplied by them. The arteries of the base of the brain are the ones that are most frequently affected, and there are secondary lesions of the parts of the brain supplied by them.

**SYMPTOMS.**—The symptoms dependent on these lesions vary in accordance with the pathological condition.

In syphilitic meningitis the principal symptoms are severe headache in various parts of the head, more or less constant, lasting for many days or even weeks, and frequently accompanied after a time by paralysis of some of the intra-cranial nerves, especially of the third or of the seventh. As in other cases of meningitis, the optic nerves may also be affected, and the child shows the general symptoms of a severe intra-cranial affection, such as vomiting and dulness.

The localized tumors or gummata present essentially the same symptoms as do the other forms of tumors of the brain in children which I have just described.

The symptoms produced by syphilitic endarteritis are the direct result of either the local dilatation or the local occlusion of the blood-vessels, which I have just mentioned. The symptoms vary according to the areas of the brain affected, but the most common ones are the various forms of paralysis of the extremities and sensory disturbances.

**DIAGNOSIS.**—In regard to the diagnosis of intra-cranial syphilis in children, the symptoms differ greatly in different cases. The most characteristic group of symptoms, and one which is exceedingly suggestive of intra-cranial syphilis, includes attacks of organic paralysis, central in origin, occurring at intervals of days or months without known cause, and without marked symptoms of either tumor or tuberculosis.

The diagnosis of cerebral meningitis may be made from the occurrence of severe headaches, followed by paralysis of one or more of the motor cranial nerves, and occurring without marked rise of temperature.

Gummata present no symptoms sufficient in themselves to distinguish them from other intra-cranial tumors, so that their existence can only be suspected.

The presence of syphilitic lesions elsewhere is our principal ground for making the diagnosis.

Syphilitic endarteritis may be suspected when an acute affection in the neighborhood of the pons or medulla not produced by traumatism occurs in a syphilitic subject, or where acute symptoms suggestive of hemorrhage or embolism occur, and where no other probable cause can be shown, such as cardiac or renal disease.



**PROGNOSIS.**—The prognosis of intra-cranial syphilis is said to be moderately favorable. The early stages of syphilitic meningitis, and sometimes even gummata, may be favorably influenced, or even cured. Of this, however, we have no decided proof, and in the more advanced cases, or where endarteritis exists, the prognosis is unfavorable, as no known remedies appear to have much influence on the secondary changes in the arteries.

**TREATMENT.**—The treatment should be with large doses of iodide of potassium, usually combined in the beginning with mercury. For a child two or three years old the initial dose of the iodide may be 0.3 gramme (5 grains) three times daily, gradually increased to 0.6 gramme (10 grains) unless gastric disturbance occurs.

**IDIOCY.**—By the term idiocy is meant a condition of marked mental deficiency. This mental deficiency may be of different grades.

**PATHOLOGY.**—Idiocy is, as a rule, the result of imperfect or impeded brain development, or it may be caused by actual destruction of portions of the brain. This condition may be produced by (1) traumatism, (2) non-traumatic inflammation, and (3) mechanical pressure.

(1) Traumatism acts usually by causing hemorrhage or destruction of the cerebral tissue in other ways.

(2) The most common form of inflammation causing idiocy is a more or less diffuse encephalitis, which ends in sclerosis and meningo-encephalitis.

(3) Hydrocephalus appears to cause or to accompany certain cases of idiocy. In some of these cases the distended ventricles cause atrophy of the cerebral tissue by pressure, while probably the distention of the ventricles is sometimes secondary. How far the degenerative conditions are primary and how far they follow pre-existing inflammations is at present unsettled.

The result of these pathological conditions is usually atrophy. This atrophy may be of intra- or extra-uterine origin, and may be local or general.

**SYMPTOMS.**—The symptoms of idiocy vary according as the individual represents a high or a low grade of this condition. An idiot may have a large head from hydrocephalus, or he may have a small head from cerebral non-development or from cerebral atrophy. Again, idiots may have normally developed crania both as to size and as to shape. In the lower grades there is often some physical malformation in connection with the mental impairment. In the more severe cases of idiocy there is considerable incoördination of the limbs, and the movements of the child are awkward and irregular. In many cases the speech is almost unintelligible. The idiot does not take notice of surrounding objects as does the normal child, and even when the sight and hearing are perfectly normal the impressions made on the senses are deadened. Epileptiform convulsions very commonly accompany idiocy, and play a most important part in the general condition of the patient.

The symptoms which are usually met with, and which enable us to

diagnosicate a pronounced case of idiocy, are the vacant expression, the occasional presence of strabismus, the drooping head, the drooling, and the lack of all idea of cleanliness. The teeth are usually decayed. Sometimes the child is so limp that he is unable to bear his weight at all, or will stand held by his parent's hands, with his feet far apart, his knees bent, and his trunk leaning forward. The whole body sways to and fro with an oscillating movement and absence of equilibrium. When able to walk alone he walks in a staggering, uncertain way, and falls easily. In many cases, however, the child cannot even sit up alone. The muscles of the neck are often so weak that the head falls over on one shoulder or forward on his chest. The vertebral column fails to support the trunk and bends to a marked degree, and all the muscles are feeble and comparatively useless. Lack of the power of attention and lack of memory exist in all cases, and in the higher grades are often the most prominent symptoms.

DIAGNOSIS.—We should be careful in very young children not to confuse slow or retarded mental development with idiocy. There is so much variation in the time at which children walk and talk, that a delayed development of these functions must not be considered to represent a condition of mental impairment. Some children develop so slowly, both bodily and mentally, that they appear very backward in comparison with others of the same age. Children in the first year of their lives may be so seriously affected by some grave disease that their development is prevented from advancing normally, and in comparison with other children of the same age they may be far below the usual grade of intelligence. If, however, we examine this class of cases carefully, we see that, although they are very backward in their development, they are gradually developing, and that they do not represent the condition of complete arrest of development which exists in idiots.

It is well to remember that in rchachitis we are apt to have not only retarded mental development but a weakness of the extremities simulating paralysis. When both these conditions occur, such cases may sometimes be mistaken for idiots.

TREATMENT.—The treatment of idiots is essentially comprised under the question of their education. The education of this class of cases should be begun early, usually from the fourth to the sixth year. Much can be done to improve the various defects which exist in each individual. He can usually be taught to coördinate his movements, and by attending to his general health his physical condition can often be much improved. In many cases if convulsions are present they can be more or less controlled. Malformations or paralyses can be treated with benefit by apparatus or by operation. The best results in these cases will be attained by placing the children in institutions devoted to the training of idiots. Parents can be told that the association of their children with others who are feeble-minded is not a disadvantage, while it is often a great disadvantage for the children of sound mind in a family to be associated with one who is idiotic. In the



large majority of cases, however, they will always have to be supervised during their lives, and, in most instances, after they have advanced to a certain point they are liable to retrograde.

I have here a feeble-minded or idiotic child (Case 307), three years old.

I shall first call your attention to the child's peculiar vacant expression, and to the fact that it behaves more like an infant than like a child. Its mental does not correspond to its physical development, for it is able to walk and to use its arms and hands freely. This child, however, was not able to support its head alone during the first year of its life, and did not learn to walk until very lately. You see that there is no especially unnatural shape to its head, which has the circumference which would be normal for a child of this age.

CASE 307.



Idiocy.

This child presents the usual variations in temper which are so common in idiots. In the very severe grades the temper is apt to be happy and quiet, while in this grade, where the physical development has not been so much interfered with, we find that explosions of temper are quite frequent. The child is not able to feed itself, and, although it will probably develop into an individual of fair strength, we can have but little hope of any improvement in its mental condition. You will notice that it drools continuously.

I shall not attempt to describe the various forms of idiocy, such as are produced by hydrocephalus, cretinism, epilepsy, syphilis, acute febrile diseases, traumata, and other causes, but shall simply mention a peculiar class which is represented by microcephalus.

MICROCEPHALUS.—When the head is under a certain size it is called microcephalic. The size which is usually accepted as representing a micro-



cephalic head is from 40.5 to 43 cm. (16 to 17 inches). According to Broca, microcephalus exists where the brain weighs 1049 grammes (35 ounces) in the male, and 907 grammes (30 ounces) in the female. It is generally considered that this microcephalic condition is due to a lack of intra-cranial pressure. Together with the lack of development of the cranial bones there exists in these cases a lack of development or atrophy of the brain, which may be considered either as the cause of the lack of intra-cranial pressure or, as is still believed by some writers, as the result of the external pressure caused by a premature synostosis. Microcephalic children are feeble-minded and usually present the symptoms of a somewhat low grade of idiocy. They not infrequently show signs of want of power of the limbs. This child which I have here is an instance of this kind.

She (Case 308) is three and a half years old, and is the eldest of three children. Her parents are healthy, as are the other children. She has never spoken. She can feed herself, and she walked when she was two and a half years old. She has incontinence of urine. She has never learned anything, has a violent temper, and sometimes has nervous attacks, which are probably of an epileptiform nature. The cranium is normal in shape, except that the forehead is very narrow, with a median vertical broad ridge. The fontanelles are closed and show no depression. There are no marked prominences about the skull. She is decidedly feeble-minded, and her attention cannot be attracted or fixed readily. The eyes are apparently normal, and her teeth are in good condition. There is a condition of paresis and incoördination, but the sensation is normal. The chest measures 49.5 cm. (19½ inches), and the head 43 cm. (17 inches).

I show her to you merely on account of the small size of the head in comparison with the hydrocephalic heads of which I have already spoken.

The treatment of these cases by craniectomy has up to the present time not proved satisfactory.

**MIRROR WRITING.**—An unusual and somewhat striking symptom which at times occurs in severe and, as a rule, chronic cerebral disease is one which is called “mirror writing.” This symptom is usually found where there is cerebral degeneration or among the feeble-minded. The actual pathology of the affection has not yet been determined. Through the kindness of Dr. Acker, of Washington, I am enabled to describe to you two cases (Cases 309, 310) of this kind which I had an opportunity of examining with him, and I shall quote freely from what he said after carefully studying these cases.

The condition represented by cases of this kind is designated “mirror writing” because the individual writes in such a way that the letters can be deciphered only when they are reflected in a mirror, when they assume the appearance of ordinary writing. These specimens of writing are similar to those which appear on blotting-paper on which the impression of an ordinary specimen of writing has been taken. The affection is usually found among left-handed children and in adults after right-handed paralysis. There seems to be a physiological tendency for left-handed children to fall into the habit of “mirror writing.” The tendency of the left hand to write in this way is, according to Erlenmeyer, due to the fact that it is easier to

use the arms in a centrifugal direction, the left from the right and the right from the left. Leonardo da Vinci was a noticeable example of this affection. The earliest recorded case of "mirror writing" was in 1688, in an epileptic girl twenty-one years of age.

Dr. Acker's first case (Case 309) was a mulatto boy, ten years of age. He was born prematurely at about the eighth month. His father is a nervous man, and does not talk plainly, but is well educated. His mother has tuberculosis of the lungs. One maternal uncle was insane.

For the first few weeks of his life he was in a very feeble condition, but finally he became healthy and strong. Whenever he was slightly sick he would have convulsions. When he was two and a half years old he fell a distance of 420 em. (14 feet) upon a bed of concrete. A deep wound in the frontal region was caused by the accident, but there was no fracture. He did not lose consciousness, and immediately after the fall responded intelligently to any questions that were put to him, but he did not cry even when the stitches were put in the cut. From the time of the accident the convulsions became more severe and more frequent. Three years ago he began to have chorea. His intelligence is about the same as that of the average child. At one time it seemed as though he would develop into a kleptomania, but at present he shows this disposition at intervals only. He is of a mild and docile temperament, has very little to say, and responds usually by a nod of the head. He is naturally left-handed, and his first attempts at writing resulted in this form of mirror writing. He has also been taught to use his right hand, and he now writes with equal dexterity in two ways with each hand.

FIG. 96.

Mirror writing of a boy 10 years old.

Here is a specimen (Fig. 96) of this boy's writing, and if you will hold it in front of the mirror you will see that it represents a child's writing, the upper line being "All nature languid seems and sad."

The next case (Case 310) was a colored boy, nine years old. His father and mother

were healthy, but of a low order of intelligence. He had two sisters who were fairly intelligent, and a brother eighteen years old who was idiotic. The boy himself was not bright, and his mother could not trust him away from home. He did not talk plainly. He had convulsions during the first year of his life, but was considered to be in fair health. He had always been left-handed, and writes "mirror writing" only.

FIG. 97.

Monkeys live in the  
forests in warm  
countries. They have  
long tails and  
like to swing  
from the trees.  
They are very  
strong and  
can jump high.

Mirror writing of a boy 9 years old.

Here is a specimen (Fig. 97) which represents some very poor writing of this boy's. The upper lines are "Monkeys live in the forests in warm countries."



## LECTURE XXXII.

## CORD.

MYELITIS.—POLIOMYELITIS ANTERIOR.—PARALYSIS CAUSED BY CARIES OF THE SPINE.—HEREDITARY ATAXIA (FRIEDREICH'S DISEASE).—LOCOMOTOR ATAXIA.—SYRINGOMYELIA.

**MYELITIS.**—The term myelitis denotes an inflammation of the spinal cord, whether of the gray or of the white matter. *Acute myelitis* has been used to designate an acute diffuse inflammation of both the gray and the white matter of the cord of non-traumatic origin, and is an affection almost unknown in children. Considerable confusion still exists in regard to the use of the term *transverse myelitis*, which from its derivation should be employed to designate an inflammation of the spinal cord extending transversely over the greater portion of a section of the cord. This term has, however, been employed to denote the results arising from compression of the cord, whether from injury or from caries or from tumor, although in these cases there exists considerable doubt as to whether any true inflammation exists. I shall therefore discard the term transverse myelitis.

The term *meningo-myelitis* is used to denote an inflammation of the meninges and of the spinal cord.

As *acute myelitis*, *meningo-myelitis*, and *hemorrhage* into the cord are extremely rare in early life, it does not come within my province to discuss them. I shall therefore begin by speaking of the form of myelitis represented by poliomyelitis anterior.

**POLIOMYELITIS ANTERIOR.**—The most frequent and therefore the most important disease which affects the spinal cord with a resulting paralysis in infancy and early childhood is called poliomyelitis anterior. This disease occurs most commonly in the first three years of life. It is rare in the first six months of life. It may occur in later childhood, and, very rarely, in adults. It is met with more commonly than cerebral paralysis.

The disease may be primary, in which case it is without any known cause; or it may be apparently secondary to other diseases, such as the acute exanthemata and erysipelas. Traumatism appears to be occasionally a cause of the disease. Most of the cases occur during the summer months.

**PATHOLOGY.**—The pathological condition which occurs in poliomyelitis anterior is now considered to be an acute inflammation of the cells of the anterior cornua of the spinal cord, with a resulting degeneration and atrophy of these cells. This condition may be confined to the anterior cornua, but in some cases it may involve the spinal meninges somewhat. So few post-mortem examinations of the early lesions connected with this disease have been made that we are dependent for our knowledge of it mostly on cases

which have been examined a number of months or years after the production of the initial lesion. These later pathological conditions are, however, quite characteristic. The circumference of the limb grows small in comparison with that of the opposite one, the result of an active muscular wasting and of retarded growth. The bones of the affected limbs are often shorter than those of the other side, sometimes even to the extent of several inches. In certain cases, however, the lengths of the bones seem to be but little affected, though the atrophy of the muscles may be very marked. The anterior cornua of the region affected, which is usually in either the cervical or the lumbar enlargement, are found to be greatly atrophied and many of the large motor cells to have been destroyed. According to Osler, the affected half of the cord may be considerably smaller than the other, and the anterior lateral column may show slight sclerotic changes, chiefly in the pyramidal tract. Accompanying this condition the corresponding anterior nerve-roots are found to be atrophied, and the muscles connected with the region of the cord which is affected atrophy and gradually undergo a fatty and sclerotic change.

SYMPTOMS.—The onset of the disease in the great majority of cases is acute. Its course is chronic. In the acute form the onset may be preceded for some days by fever and restlessness, but it is very apt to appear suddenly, with, at times, convulsions which, as a rule, are of a milder type than those which occur in cerebral paralysis.

In addition to the cases which are manifestly acute in their origin there have been mentioned certain subacute and chronic cases. There is some doubt, however, whether these cases do not originate in the same manner as those which are called acute. The probability is that in most of the cases which appear not to have had an acute onset and in which the paralysis seems to develop slowly, the early acute onset has been overlooked. This subacute variety of poliomyelitis anterior does not differ from the acute cases in any way in its symptoms, prognosis, diagnosis, and treatment.

Following the acute onset there are at times unconsciousness, lasting sometimes for a number of days, vomiting, general nervous disturbance of the bladder and intestines, and a variety of symptoms of nervous irritability which may represent the prodromata of a number of diseases. The temperature is seldom very high,  $38.3^{\circ}$  to  $38.7^{\circ}$  C. ( $101^{\circ}$  to  $102^{\circ}$  F.); it may, however, in certain cases be higher. At times there are no prodromata, but the paralysis is noticed in the morning after a night's rest, although on the evening before the child was seemingly perfectly well. The severity and length of the prodromal symptoms are no indications of the gravity of the lesion or of the prognosis as to recovery. Pain in the paralyzed limb is not an uncommon symptom, but occurs only very early in the disease. The disease is primarily a motor disturbance, sensation remaining intact. Cerebral symptoms, if present, pass off rapidly with the appearance of the paralysis. The paralysis is usually apt to affect more than one limb in the beginning, but, as a rule, soon becomes monoplegic. The leg is more frequently



affected than the arm. Paraplegia in the beginning is not uncommon, and all forms of paralysis may occur. There may also be diplegia, cross paralysis, the affection of both arms, and paralysis of the muscles of the back and abdomen. Hemiplegia, so common and almost characteristic of cerebral paralysis, may be present, but is rare in poliomyelitis anterior. The muscles most frequently affected are the extensors, adductors, and supinators. The distribution of the paralysis is usually in groups of muscles. The respiratory muscles may be affected, though rarely. Facial paralysis is so rare that it can almost be said never to occur in uncomplicated poliomyelitis. When the prodromal symptoms have passed off, as they usually do very quickly, the functions of the child are carried on as usual, and the general growth and mental activity are unimpaired. The tendon reflexes disappear in the affected limbs. When the paralysis has reached its height it remains stationary for perhaps from three to six weeks, and then gradual improvement begins, and goes on in certain groups of the paralyzed muscles for six or eight months, leaving other groups paralyzed. These groups again at times recover entirely or remain disorganized, and thus lead later to contractures and deformities. When contractures occur they appear later than do those of cerebral origin. These contractures are to be distinguished from those of cerebral paralysis, which are always spastic, while those of spinal origin are paralytic.

When paralysis affects wholly or chiefly the gastrocnemii and posterior tibial muscles, the other groups act predominantly, causing dorsal flexion of the foot, so that the child walks on its heel. This condition is termed *talipes calcaneus*. When, on the other hand, the *tibialis anticus* and anterior muscles of the leg are most affected, the deformity of *talipes equinus* occurs; and if the *peronei* muscles remain unaffected, there is *valgus*, while if they are affected with the anterior group, *talipes equinus varus* occurs. Dislocation of the hip may occur in certain cases of complete paralysis of the leg. Severe cases may show complete flaccidity, and not unfrequently the ligaments about the joints are so weakened that the joints become too movable, and the condition called *flail-joint* results. This condition may be present at the hip, knee, or ankle, and sometimes at the shoulder or wrist. Marked atrophy appears in a few weeks. Muscular atrophy, rapid and extreme, is the rule in poliomyelitis anterior. Shortening of the bones from arrest of growth may also appear. The surface temperature of the affected limb is lowered, the limb feels cold, relaxed, and lifeless, and the circulation is generally sluggish. Spasmodic movements, except the primary convulsions, are absent.

**DIAGNOSIS.**—In the stage of onset, and until paralysis has appeared, the diagnosis must be held in abeyance. The salient points by which a diagnosis can usually be made are (1) sudden paralysis; (2) loss of tendon reflex; (3) rapid atrophy; (4) cold, flaccid limbs; (5) absence of impairment of sensation; (6) presence of the reaction of degeneration and a diminished reaction to the faradic current.



It is always difficult to diagnosticate poliomyelitis in the initial stage of the disease. At that time pain and sensitiveness of the affected limb may be present, and may lead us to suspect that the disease is rheumatism. The convulsions and unconsciousness which may appear at this stage are so often present in other diseases that they are not of much aid in making the diagnosis of poliomyelitis anterior.

The most reliable test at our command for making the diagnosis of poliomyelitis anterior is the electrical reaction, and when this can be obtained it clearly characterizes the disease. The normal muscles react to both the faradic and the galvanic current. In applying the galvanic current a quick muscular contraction is noticed both on the opening and on the closing of the negative (cathodal) and of the positive (anodal) pole, but is greater when the cathodal pole is closed. When the galvanic current is applied to the muscles affected by poliomyelitis anterior, the contractions continue, but are slower and less sharp, and the reverse of what takes place in normal muscles occurs. Thus, the anodal closure causes a contraction equal to or greater than that caused by the cathodal closure (reaction of degeneration). As the muscles recover there is first a return to the normal galvanic reaction and later to their normal faradic excitability. The diagnosis in young children, however, by means of the galvanic current is practically useless except in the hands of an expert. The faradic excitability begins to diminish within a few days after the onset of the paralysis, and disappears entirely from those muscles which are severely affected.

DIFFERENTIAL DIAGNOSIS.—Poliomyelitis anterior is most apt to be mistaken for cerebral paralysis, and can be best differentiated from that disease by means of the symptoms which I have already described, and which are represented in this table (Table 104).

TABLE 104.

	Cerebral Paralysis.	Poliomyelitis Anterior.
Pathology . . . . .	Hemorrhage. Embolus. Thrombosis. Sclerosis. Atrophy. Porencephalia.	Inflammation of anterior cor- nua of cord.
Age . . . . .	Under three years.	Under three years.
Onset . . . . .	Acute febrile.	Acute febrile.
Motor disturbance . . . .	Paralysis. Most common form hemiplegia. Spastic rigidity. Spastic gait. All the muscles of a limb affected.	Paralysis. Most common form monoplegia. Flaccid. Groups of muscles in a limb affected, usually the exten- sors.
Contractures . . . . .	Of all the muscles, especially the flexors and adductors.	Of the flexors in the calf.

TABLE 104.—*Continued.*

	Cerebral Paralysis.	Poliomyelitis Anterior.
Spasmodic movements . . . . .	Athetosis. Post-paralytic chorea. Epileptiform convulsions.	Absent. Convulsions may occur at the onset of the disease.
Sensation . . . . .	Unaffected.	Unaffected.
Nutrition . . . . .	Arrest of growth.	Tendency to extreme atrophy coming on early in the paralyzed limb.
Electrical reaction . . . . .	Normal.	Reaction of degeneration.
Tendon reflex . . . . .	Exaggerated on the paralyzed side.	Absent.
Speech . . . . .	Liable to be impaired.	Unimpaired.
Intelligence . . . . .	Often impaired.	Normal

Other affections which may be mistaken for poliomyelitis anterior are (1) the paralysis following multiple neuritis; (2) progressive muscular atrophy; (3) pseudo-hypertrophic muscular paralysis; (4) rhachitic pseudo-paralysis; (5) scorbutus.

(1) The principal points by which multiple neuritis is to be distinguished from poliomyelitis anterior are (*a*) the symmetrical affection of the limbs in the former and tenderness over the nerve-trunks; (*b*) the atrophy in multiple neuritis is not so severe as in cases of poliomyelitis anterior; (*c*) the course of the disease is different, cases of multiple neuritis almost invariably recovering, while severe cases of poliomyelitis do not recover.

(2) Progressive muscular atrophy, to which I shall refer in a later lecture (page 763), is so rare an affection among children that it need only be alluded to. There have been a few cases, however, where this disease began in children in the legs, and the paralysis is to be distinguished from that of poliomyelitis by its gradual onset, by the galvanic reaction continuing normal, and by the faradic excitability usually remaining as long as there is any muscular substance left. In this disease, also, the reflexes are not lost until the muscular substance has disappeared.

(3) Pseudo-hypertrophic muscular paralysis in its early stage is not likely to be mistaken for poliomyelitis, for the absence of abnormal electrical reaction, the increase in the size of the muscles, and its gradual onset are distinguishing points; although in the later stages of this disease atrophy may occur, the history will then clearly differentiate the condition from poliomyelitis.

(4) In certain cases of rhachitis the power of using the legs is so much affected that the mistake is quite commonly made of supposing that these

children are affected by poliomyelitis anterior. The condition in rachitic children is one of weakness and not of paralysis, and can be distinguished by the normal electrical reaction of the muscles and the lack of pronounced atrophy.

(5) The pseudo-paralysis which is commonly seen in cases of scorbutus is often mistaken for some organic disease of the central nervous system, with its resulting paralysis. The differential diagnosis from poliomyelitis anterior, however, is not difficult to make, for the involvement of other joints in addition to those of the legs, the presence of pain and tenderness to such a degree that the child cries whenever the limbs are touched, and the normal temperature of the skin clearly distinguish this condition from poliomyelitis, in which disease normal sensation, freedom from pain, and a cold feeling of the limb affected are found.

PROGNOSIS.—So far as a fatal issue is concerned, the prognosis is very favorable. If death occurs it usually takes place at the end of one or two weeks, and is the result of interference with respiration, which may be caused where the paralysis is extensive. Where in the initial stage of the attack cerebral symptoms are prominent and continue for some time, the prognosis is grave.

A second attack of the disease is very rare, and when it occurs it usually comes a few days after the original attack, and manifests itself by an increase of the existing paralysis. The paralysis, as a rule, will not increase when it has been stationary for twenty-four hours. The tendency of poliomyelitis is for a time to improve. Some of the limbs affected recover in the first few days, but in those which remain affected longer perfect recovery is rare. When there is no improvement after six or eight months the probability is that entire recovery will never take place, though under proper treatment a slight improvement may go on for years.

We must remember that, even when untreated, a case of poliomyelitis is very apt to improve for one or two months quite rapidly, then rather slowly for two or three months, and then, after a stationary period, contractions, looseness of the joints, and malpositions may begin to be evident and may increase indefinitely.

When proper treatment is carried out, the prognosis is much more favorable, and the period of possible improvement can be extended for some years. According to Bradford and Lovett, there is certainly no leg, however wasted and contracted, that is not amenable to some improvement by operative or mechanical treatment.

TREATMENT.—The treatment of poliomyelitis by means of drugs has produced such unsatisfactory results that it may be said to be useless. The very multiplicity of the remedies which have been experimented with proves their inefficiency. It is doubtful whether any treatment by drugs can be beneficial to a central lesion of this character. Although a number of remedies have been recommended to be given in the onset of the attack, it is probable that none of them are of any especial benefit; though it is wise to



see that the bowels are freely moved, and, if the attack has been ushered in by convulsions, to treat these symptomatically if they continue.

Although we know of no rational means of treating the primary lesion of poliomyelitis anterior, we know that the results of this lesion, as shown by paralysis of the muscles, are such that the paralysis should be treated at once. The indication is to combat the rapid atrophy which is part of the disease, and to prevent its increase, and its later results from proceeding to a degree which would interfere with subsequent repair. To accomplish this, the affected limb should be supported in a normal position and carefully guarded against the stretching of joints, ligaments, and muscles. In addition to this, gentle massage and the faradic current applied five or ten minutes at a time at least four or five times a week are indicated to keep the affected muscles in the best possible condition and to combat the atrophy which to a greater or less degree occurs. The regular application of heat is also found to be useful where the limb is cold. According to Bradford and Lovett, muscles are much less likely to contract and deformities thus less apt to result in properly supported limbs.

The later manifestations of club-foot and other deformities should be dealt with by the orthopædic surgeon.

It may be well to mention that I have adopted the name poliomyelitis anterior as best representing the disease as we now know it. It has been called with less reason by various names, such as *infantile paralysis*, *essential paralysis of children*, *acute atrophic spinal paralysis*, *myelitis of the anterior horns*, *myogenic paralysis*, *dental paralysis*, and *poliomyelitis anterior acuta*.

I have some cases here such as you will be likely to meet with in your practice, and I shall now examine them before you.

This little girl (Case 311, page 683) is nine years old.

She was perfectly well up to the time of an attack, which came on suddenly and without known cause. She is said to have fallen while she was playing, but no injury of the leg could be detected, though she was carefully examined under ether. The exact date of the attack is not known, but it was some time ago. Her general health is reported to have been very good, and she seems to be bright mentally. She is, as you see, well developed, and has a good color. Nothing abnormal has been detected on physical examination of the lungs, thorax, abdomen, or other organs. The pulse is regular and of good strength. The left leg shows considerable atrophy, being 4.37 cm. ( $1\frac{3}{4}$  inches) smaller than the right in the calf and 2.5 cm. (1 inch) in the thigh. The leg is somewhat cyanotic, and is cold to the touch. There is marked weakness of the muscles below the knee, especially the extensors of the foot and toes. When she is lying in bed the movements of the thigh are performed with some strength. On walking she rotates the leg outward, so that the foot is at right angles with the line of motion, and she drags the toes. The joints are freely movable. Nothing abnormal has been detected in connection with the spine, which presents the condition of a movable lateral curvature, due to the shortening of the affected leg.

She is being treated by massage, electricity, applications of hot cloths twice daily for half an hour, and by apparatus.

She represents the condition of poliomyelitis anterior of the left leg, and, although she may receive some slight benefit from treatment, the probability is that she will always be lame.

This boy (Case 312) is twelve years old.

He is said to have had rheumatic fever when he was thirteen months old. It was noticed that he dragged his right leg when creeping, and this leg has evidently been affected ever since he began to walk. The leg is atrophied, and there is a condition of valgus in the foot. There is slight permanent flexion in the knee, and the hip is also slightly flexed.

CASE 311.



Poliomyelitis anterior. Left leg. Female, 9 years old.

The movements of the limb are otherwise good. The adductors are in good condition. The abduction is chiefly accomplished by means of the tensor vaginæ femoris.

This case is evidently one of poliomyelitis anterior, with valgus of the right foot.

This infant (Case 313, page 684), twenty months old, is an unusual and interesting case of infantile paralysis of the abdominal muscles.

He is stated to have always been healthy until five weeks ago, when on coming into the house he vomited and two days later limped a little. When the infant cries you see that the abdominal walls bulge, especially on the left side. The motion of the left leg is very free, but slightly flaccid. The patellar reflex is absent, and he sits up very feebly.

This little girl (Case 314, page 684) is two and one-half years old, and presents the same condition as the previous case.

She has a sister who is said to have had an attack of poliomyelitis anterior when she was ten months old. No other history has been obtained about this case, except that she was well and strong until this attack, which occurred six weeks ago. The onset of the dis-



ease was sudden, and was accompanied by high fever, followed in three days by complete paralysis of the muscles of the upper and lower extremities of the body and of the head. The arms and head soon recovered. She is unable to sit up alone, and the abdominal muscles are paralyzed to such an extent on the left side that, as you see, they are flaccid, bulge out,

CASE 313.



Poliomyelitis anterior. Abdominal muscles, left side. Male, 20 months old.

CASE 314.



Poliomyelitis anterior. Abdominal muscles, left side. Female, 2½ years old.

and do not react normally. The left leg is perfectly flaccid. The knee-jerks are absent. The surface temperature is diminished, and there is atrophy of the legs.

(Subsequent history.) Under treatment with electricity and massage, complete recovery took place.

The next case (Case 315, page 685) is that of a boy (I.), six and one-half years old, who was apparently healthy at birth, and who has never had any illness.

When he was one year old he was noticed to have some motor disturbance of the left leg. On examination of the leg the surface temperature is found to be diminished, the knee-jerk is absent, and there is an atrophy of 5 cm. (2 inches) of the thigh and 6.5 cm. (2½ inches) of the calf. There is also 3.7 cm. (1½ inches) shortening in the leg. The child walks, as you see (II.), with a marked limp of the left leg, and there is the condition of flail-joint in his left knee and ankle.

These symptoms, without any further history of the case, justify us in making a diagnosis of disease of the spinal cord rather than of the brain. This is a typical case of the appearances presented in the advanced stages of a severe case of poliomyelitis anterior.



This girl (Case 316, page 686), sixteen years old, represents very well the results which may occur from an attack of poliomyelitis anterior.

She is said to have had some disturbance in her left leg following a fall from a high chair when she was seven months old. She did not walk until she was eight years old, and has been lame ever since. She came under my observation at the hospital when she was thirteen years old, and at that time presented the evidences of a long-standing paralysis of spinal origin. The knee-jerk was absent. The left leg was cold and atrophied, and the

## CASE 315.

I.



II.



Poliomyelitis anterior. Flail leg, left side. Male, 6½ years old.

foot was in the valgus position. She has, you see, a lateral curvature, due to paralysis of the muscles of one side of the back. She has shown only slight improvement under treatment for the last three years.

Here is a little girl (Case 317, page 686), five years old, who, as you see, has paralysis of both legs.

She was well and strong until about her third year, when she had an attack of whooping-cough. During this attack she also had some other illness, which was characterized by fever and pain in the back. The loss of power of her legs dates from this time, and is said to have been gradual. She is fairly well developed, and the paralysis has affected both legs and thighs as well as the psoas and iliac muscles. There is marked atrophy, and the reflexes are absent.

You notice in this case that the limbs are held apart and are flaccid. If the case were one of cerebral paralysis there would be in place of this flaccid condition a contraction of the adductors of the thigh, which would have been apt to hold the limbs closely together.

At times this contraction would perhaps be so strong as to prevent the limbs from being drawn apart.

The prognosis for complete recovery in this case is unfavorable.

The treatment will be of a general nature, such as I have already explained to you should be adopted in cases of this class.

CASE 316.



Poliomyelitis anterior. Talipes varus. Lateral curvature. Female, 16 years old.

CASE 317.



Poliomyelitis anterior. Paralysis of both legs. Female, 5 years old.

This little girl (Case 318, page 687), five years old, is a case of poliomyelitis anterior which has affected the right leg.

When she was three years old she fell from a step, and was seized with a sudden attack of paralysis of the right leg. A month later she walked with toe-drop of the right foot and slightly of the left. The skin of the limb is not especially cold or blue. The right thigh measures 24.1 cm. (about  $9\frac{1}{4}$  inches), the left thigh 24.3 cm. ( $9\frac{3}{4}$  inches). The right calf measures 16.2 cm. ( $6\frac{1}{2}$  inches), the left 17.5 cm. (7 inches). The patellar reflex is absent on the right side and very slight on the left. The right leg is 1.2 cm. ( $\frac{1}{2}$  inch) shorter than the left.

Under the application of massage and the use of various mechanical apparatus there has been slight improvement.

This boy (Case 319, page 687), eleven and one-half years old, has a good family history,



and is said to have been swung about by his feet when he was seven months old, to which the family attribute the present condition of his right foot.

The anterior portion of the foot is flexed, as you see, at a sharp angle at the mediotarsal joint. The foot can be easily bent to a right angle, but not beyond. Tense bands of plantar fasciæ can be felt when the foot is straightened out, but it can be brought into position by the use of considerable force. The length of the legs is equal. There is 1 cm. (about  $\frac{3}{8}$  inch) atrophy in the right calf and 0.6 cm. ( $\frac{1}{4}$  inch) of the right thigh.

CASE 318.



Poliomyelitis anterior. Paralysis of right leg. Female, 5 years old.

CASE 319.



Poliomyelitis anterior. Talipes equinus on right side. Male, 11½ years old.

He represents the condition of talipes equinus, the result of a contraction of the flexor muscles following an attack of infantile paralysis.

I have also here a boy (Case 320), twelve years old, who illustrates a case of poliomyelitis anterior secondary to erysipelas.

He had an attack of erysipelas when he was fourteen months old. The erysipelas lasted for about one month, and was followed by an attack of diarrhœa which lasted for six weeks. It was noticed that the infant was weak and had little power in the left leg about one week after the beginning of the erysipelas. After recovering from the diarrhœa he began to walk a little, but with a limp, which he has had ever since. The leg has since been growing smaller, and he has lately shown no improvement whatever. There has never been any pain in the leg. He walks with a decided limp, and the foot is brought to the floor with a slap. The knee bends backward beyond its proper position. The leg is



much atrophied, the right thigh being 11.2 cm. ( $4\frac{1}{2}$  inches) less than the left, and the leg 7.5 cm. (3 inches) less than the left leg. The leg and foot of the affected limb are slightly colder to the touch than those of the other. On raising the foot of the affected limb it is seen that hyperextension can be produced to an angle of  $140^{\circ}$ .

In this case I shall advise apparatus to prevent the further formation of flail-joint at the knee, which is evidently now present.

**PARALYSIS CAUSED BY CARIES OF THE SPINE.**—In cases of paralysis caused by caries of the spine the lesion is essentially a compression of the cord: this is usually slow in its progress, and it is doubtful whether in it a true inflammation occurs even in the beginning. The condition resulting from compression occurring in the course of caries of the spine may be found in any part of the cord. It is most frequently met with in disease of the dorsal region, though it may occur in the cervical and lumbar regions. In caries of the spine the compression of the cord is not often the result of pressure from the vertebræ, but usually is caused either by an abscess in the vicinity of the diseased vertebræ, or more commonly by a thickening of the meninges.

When the lesions of the cord are of any considerable extent, ascending and descending secondary degenerations follow after a time. If the process ceases, it leaves a certain amount of sclerosis of the cord at the seat of the disease. This may be very slight, or the cord may be considerably reduced in size and yet may transmit normal nervous influences.

**SYMPTOMS.**—The onset of the disease is sometimes quite sudden, but more frequently it is rather gradual. The symptoms vary according to the part of the cord which is affected.

When the lesion is in the dorsal or the lumbar region the onset is represented by numbness and weakness in the legs. This is quickly followed by a paralysis which may become complete in a short time.

If the lesion is below the level of the sixth dorsal vertebra, the legs alone are affected; if on a level with this point, the abdominal muscles are involved. Sensation up to nearly the level of the lesion may be diminished, or even lost entirely. In regions above the lumbar enlargement the reflex reactions are exaggerated and ankle-clonus soon appears.

When the disease affects the cervical enlargement, or any portion of the cord above, all the extremities are apt to be paralyzed. In severe cases there will be retention of urine, with subsequent incontinence. The bowels are usually constipated, but incontinence of fæces is sometimes present.

In lesions of the lumbar enlargement the knee-jerks will be lost. Trophic changes in the limbs are not marked, but the muscles are somewhat wasted, and rigidity may develop. Bed-sores are apt to form. The reaction of degeneration is not present.

The characteristic picture of lesions in the dorsal region caused by caries of the spine is paraplegia.

**DIAGNOSIS.**—The disease is to be differentiated from poliomyelitis anterior, in which disease monoplegia is more common than paraplegia, and in

which the reflexes are lost and the action of degeneration is present. In addition to this means of making a differential diagnosis, the absence of initial fever and prodromata, of disturbances of sensibility, of paralysis of the sphincters, and of a tendency to bed-sores in poliomyelitis anterior is of great aid in differentiating it from the results of injuries of the spine, where rigidity of the limbs, increased reflexes, and contractures are prominent symptoms.

The differential diagnosis from cerebral paralysis is more difficult, as the condition of the limbs is similar in both. The diagnosis is made by the absence of all cerebral symptoms, and by the presence of such special symptoms as rigidity and prominence of the vertebræ in injuries of the spine.

**PROGNOSIS.**—The prognosis in these cases is in general favorable. A certain number of cases remain un cured, but nearly all recover under treatment, although the condition may persist for months.

**TREATMENT.**—The treatment of these cases is, as a rule, to be directed to the injuries, and consists essentially in perfect rest on a rectangular bed-frame. Massage and electricity are sometimes of assistance when applied to the paralyzed limbs. Where no improvement occurs after several months, laminectomy must be considered; and there has been a case (Case 321) at the Children's Hospital where improvement followed this operation. In this instance an abscess was pressing upon the cord, and on its being discovered and emptied recovery took place. The operation was performed by Dr. H. L. Burrell.

**HEREDITARY ATAXIA** (Friedreich's Disease).—Hereditary ataxia is a very rare disease. It is an organic affection of the spinal cord, usually occurring in several members of a family and developing in later childhood. The names family ataxia and generic ataxia have also been used.

**PATHOLOGY.**—The pathology of the affection is a slow, progressive degeneration of the posterior and lateral columns of the cord.

**SYMPTOMS.**—The characteristics of this disease are its slow development, staggering gait, loss of muscular power, nystagmus, sometimes loss of knee-jerk, frequent disturbance of speech, and finally complete helplessness with mental impairment.

**PROGNOSIS AND TREATMENT.**—The prognosis of hereditary ataxia is always unfavorable, and there is no known remedy which is of benefit.

**LOCOMOTOR ATAXIA.**—In connection with this degeneration of the posterior and lateral columns of the cord which occurs in hereditary ataxia, I shall merely mention the degeneration of the posterior columns of the cord (locomotor ataxia), as this disease is almost unknown in childhood. The disease as it occurs in children usually involves the lateral as well as the posterior column of the cord, and is thus closely related to Friedreich's disease.

Locomotor ataxia is to be distinguished from multiple neuritis, which it sometimes closely resembles, the pain, ataxia, and loss of knee-jerk often occurring in both. The diagnosis from multiple neuritis is to be made

chiefly by the presence of ocular symptoms in locomotor ataxia, such as the Argyll-Robertson pupil. (In this condition the pupil does not react to light, but does react to accommodation.) In addition to this means of differential diagnosis, the tenderness of the nerve-trunks in multiple neuritis does not occur in locomotor ataxia. You must also remember that locomotor ataxia is incurable, while multiple neuritis always recovers.

Locomotor ataxia may be differentiated from Friedreich's ataxia by (1) the fact that it is not of hereditary origin, (2) the absence of nystagmus and of mental symptoms, and (3) the ataxic and shuffling gait.

**SYRINGOMYELIA.**—As defined by Osler, syringomyelia is a gliomatous new formation about the central canal of the spinal cord, with cavity formation. This disease is so rare in children that I shall merely state that it is now regarded as a gliosis, a development of embryonal neuroglial tissue in which hemorrhage or degeneration takes place with the formation of cavities.

In this disease we usually find a diminution of sensation to heat and cold, according to the site of the lesion, which is commonly a point in the upper dorsal or the lower cervical region. There is apt to be a weakness of one or both arms, accompanied by marked wasting. There is also usually some weakness in the legs. The reflexes are increased, and a spastic condition is likely to result. These symptoms are usually accompanied by marked lateral scoliosis.

In typical cases the diagnosis is easily made where there is an amyotrophic paralysis of one or both of the upper extremities, with retention of tactile sensation and loss of thermic and painful sensation below the dorsal region, and a weakness of the lower extremities, with a tendency to spastic rigidity.

Syringomyelia is an incurable disease, and the treatment is therefore usually limited to correcting, if possible, the lateral curvature which frequently accompanies it.



## LECTURE XXXIII.

## BRAIN AND CORD.

## MULTIPLE CEREBRO-SPINAL SCLEROSIS.—CEREBRO-SPINAL MENINGITIS.

**MULTIPLE CEREBRO-SPINAL SCLEROSIS.**—By multiple sclerosis of the brain and cord we mean a disease in which the nerve-elements of certain areas in the brain and cord are more or less replaced by connective tissue. The sclerosis which occurs in these cases, however, is not a distinctive lesion of multiple cerebro-spinal sclerosis, as it is the same that occurs in other sclerotic conditions of the nervous system. It is simply the multiple distribution of these areas which is pathognomonic of the disease. The disease has also been described under the name of disseminated sclerosis, insular sclerosis, focal sclerosis, herdisklerose, and sclérose en plaques.

**ETIOLOGY.**—The etiology of the disease is obscure; but heredity appears to be one of the causes of multiple sclerosis, and traumatism, shock, and various acute diseases, especially those of an infectious character, have an etiological significance.

**PATHOLOGY.**—Only a small number of autopsies of this disease have been made in children.

The characteristic feature of the disease by which it is distinguished from other sclerotic diseases of the brain and cord is the erratic and multiple distribution of the sclerosis. The sclerotic patches may occur in the brain or in the cord, or in both, and they are perfectly irregular as to the parts of the cerebro-spinal system which they involve. According to Osler, there is an increase in connective tissue of the sclerosed patches, and their fibres are denser and firmer than normal. The gradual growth destroys the medulla of the nerves, but the axis cylinders persist in a remarkable way.

**SYMPTOMS.**—The onset of the disease may be rapid or slow, but is more likely to be rapid. According to Pritchard, the child is noticed, after perhaps a blow on the head, or a shock or fright, or without any apparent cause, to tremble. In some cases the disease may be ushered in by a convulsion. In connection with the tremor, nystagmus may appear as an early symptom, but, as a rule, it is a later one. The gait is usually affected early, the movements being clumsy or staggering. Among other early symptoms strabismus and diplopia may be mentioned. Headache and vertigo are probably not infrequent, although in young children it is somewhat difficult to determine the presence of these symptoms. Exaggeration of the reflexes which depend upon the location of the lesion is an early symptom in some cases, and may be associated with ankle-clonus. The later symptoms are disturbance of speech, mental weakness, slow muscular wasting, and paralysis of the extremities.

**DIAGNOSIS.**—The differential diagnosis of multiple cerebro-spinal sclerosis is to be made chiefly from hereditary ataxia, as there is no other disease of the nervous system occurring in children which especially simulates it. Although in both diseases ataxia, nystagmus, and defects of speech occur, and although tremor is a common symptom of both, yet these symptoms differ somewhat in their form.

According to Pritchard, *tremor* is a common symptom in multiple cerebro-spinal sclerosis in children, while in hereditary ataxia it occurs in only a certain proportion of cases ; in the former disease it is of the voluntary type, in the latter it is of the choreiform variety. Again, in hereditary ataxia the affection of the *speech* occurs, as a rule, later than in sclerosis. On the other hand, *ataxia* of the extremities is less constant in sclerosis than in hereditary ataxia, and the *inability to stand* with the feet together and the eyes closed, while common in hereditary ataxia, is rarely observed in sclerosis. Various paræsthesiæ which not infrequently occur in hereditary ataxia, especially the *girdle sensation*, are not common in children affected with sclerosis.

In addition to these other clinical differences there are three symptoms which afford a marked contrast in the two diseases. These are (1) the condition of the reflexes, especially that of the patellar tendon ; (2) the mental state ; and (3) the tendency to convulsive seizures. In multiple sclerosis the knee-jerk is commonly exaggerated and rarely abolished, while in hereditary ataxia it is often abolished. The mental condition is commonly dulled at some stage of the disease in multiple sclerosis, and is usually in the form of a simple dementia. In hereditary ataxia, on the contrary, the intellect is unimpaired, mental weakness being exceptional. Convulsions are quite common in sclerosis and are rare in hereditary ataxia.

A differential diagnosis should also be made from chorea, which can be eliminated readily by the absence of tremor, by the presence of incoördinate movements, and by the absence of nystagmus and of true ataxia.

**PROGNOSIS.**—The prognosis in multiple cerebro-spinal sclerosis for permanent recovery is very unfavorable. The disease may be arrested temporarily, but improvement in the general condition of the child, as a rule, merely marks a remission. The child rapidly becomes so helpless that there is a corresponding liability to complications and to death.

**TREATMENT.**—There is no drug which appears to have any effect upon the disease, the treatment being wholly symptomatic. The general health of the child should be carefully attended to, and in this way the inroads of the disease on the nervous system can be combated.

**CEREBRO-SPINAL MENINGITIS.**—By cerebro-spinal meningitis we mean an acute infectious disease characterized by a leptomeningitis of the brain and spinal cord.

Although this disease is usually classed under the infectious fevers, it seems to me to be associated more naturally with diseases of the nervous system, because the main pathological lesions are found in the brain and in



the spinal cord. However closely it may in the future be proved to be associated with other diseases, such as pneumonia, and however firmly we may believe that its cause is a microbe as in the other diseases of the infectious class, still the salient symptoms by which we can make our diagnosis are those produced by central organic nervous lesions. The disease does not appear to be contagious. It may be acute or chronic. It may occur as a primary disease or in connection with some other infectious disease, such as acute lobar pneumonia.

ETIOLOGY.—Cerebro-spinal meningitis at times occurs in wide-spread epidemics. It is also met with in a sporadic form. Although there has not yet been made a sufficient study of the epidemic form of the disease to allow me to state much that is definite about its causation, it is probable that it is the same as in the sporadic form. From the sporadic cases which have been carefully studied it is evident that certain bacteria are the cause of the disease. The most common organism which has been found is the pneumococcus of Fraenkel, but the streptococcus and staphylococcus pyogenes aureus have also been found in a few cases. No distinction except a bacteriological one can be made between the cases in which these bacteria are found; nor can any be made between the epidemic and the sporadic cases, as they have the same symptoms. Although there is supposed to be a close connection between cerebro-spinal meningitis and pneumonia, yet the former disease is frequently found without the lesions of pneumonia being present.

PATHOLOGY.—The pathological lesions which represent this microbic form of cerebro-spinal disease are chiefly an inflammation of the pia mater, with its accompanying serous, fibrinous, or purulent exudation. The brain and cord may be involved. Foci of hemorrhage and of encephalitis are sometimes found. The prominent primary lesion which produces the typical, uncomplicated picture of the acute variety of cerebro-spinal meningitis in its early stage is a leptomeningitis, and the disease can well be looked upon as a *microbic leptomeningitis*.

As has been well stated by Delafield and Prudden, the degree of the lesion in the brain varies greatly, depending upon the period at which death occurs. At times, when death occurs early in the disease, no macroscopic change will be evident. Microscopic examination in these cases, however, shows a moderate degree of extravasation of leucocytes in the vicinity of the vessels. In well-marked cases the pia mater and the cord are more or less densely infiltrated with serum, fibrin, and pus. This pathological condition may be found over the convexity and base of the brain, and is frequently most marked in the latter situation. In the cord the infiltration may occur over the anterior and posterior surface, and in some cases, probably owing to the recumbent position of the patient, it is most marked on the posterior surface. The ventricles of the brain and the central canal of the cord may contain turbid serum mingled with pus-cells and sometimes blood-cells. The membranes and underlying nervous tissue may be hyper-



æmic and the seat of capillary hemorrhages. In protracted cases the ventricles may be dilated with serum.

In addition to these characteristic lesions of the disease, there are a number of secondary changes in different parts of the body, which are not constant, but which occur with sufficient frequency to warrant their mention. Thus, there may be subserous punctate hemorrhage in the endocardium; petechiæ in the skin; hyaline and granular degeneration in the voluntary striated muscle; occasional multiple abscesses in various parts of the body; suppurative inflammation of the joints; parenchymatous degeneration of the heart, liver, and kidneys; swelling of the gastro-enteric lymphatic system, and metastatic choroiditis.

The lesions are essentially the same in the epidemic and sporadic cases of acute cerebro-spinal meningitis.

In the form which from its length may be called chronic the pathology has not been determined, as a sufficient number of autopsies of this variety has not yet been obtained. It is, however, possible that the various later symptoms of organic central disease which occur in some of these cases, and especially in those which do not recover, may be produced by the lesions of hydrocephalus and cerebral abscess.

**SYMPTOMS.**—The disease is usually sudden in its onset, attacking at times perfectly healthy children. The prominent symptoms are intense headache, photophobia, and at times convulsions, pain, hyperæsthesia, vomiting, delirium, and, later, coma; also sensitiveness to sound and to touch. Tenderness on pressure over some portion of the vertebral column is found not uncommonly. The temperature in the more severe cases is high,  $40.6^{\circ}$  to  $41.1^{\circ}$  C. ( $105^{\circ}$  to  $106^{\circ}$  F.); usually, however, it is  $38.3^{\circ}$  to  $38.9^{\circ}$  C. ( $101^{\circ}$  to  $102^{\circ}$  F.). There is no regular temperature curve; in fact, the symptoms, temperature, pulse, and respiration vary in different cases. The pulse is usually quick; the respirations are rhythmical, but somewhat quickened. The bowels are usually constipated.

Strabismus is a common symptom, and rigidity and retraction of the neck and back (opisthotonos) are soon noticed. The knees are usually drawn up. The child emaciates rapidly. The pupils are altered. It is not uncommon to find metastatic choroiditis with exudation of pus into the vitreous (Wadsworth). There is often bilateral loss of hearing. Remissions in the symptoms are frequent. A *tache cérébrale* can at times be found. The spleen, especially in acute cases, is enlarged. If the brain and cord are also decidedly involved, symptoms corresponding to the locality and degree of the lesion appear. This is especially noticeable in the chronic form, where the disease has lasted for some months. Pneumonia, arthritis, pleuritis, and pericarditis may arise as complications.

**DIAGNOSIS.**—The prominent symptoms on which you must rely in differentiating cerebro-spinal meningitis from tubercular meningitis, for which it would be most likely to be mistaken, are the sudden onset, extreme headache and hyperæsthesia, opisthotonos, herpes, and regular pulse in the

cerebro-spinal disease as compared with the usually slower progress and milder symptoms of the tubercular. In some cases the onset is not so sudden, and difficulties have arisen in the differentiation from typhoid fever and pneumonia; but, except in the rather rare meningeal types of these latter diseases, the diagnosis will in a few days become clear.

In young infants the symptoms of cerebro-spinal meningitis may be merely a heightened temperature with clonic convulsions, so that the diagnosis cannot be made during life from the various forms of reflex convulsions which may occur at this age, and cerebro-spinal meningitis can only be suspected. A case illustrating this fact was seen by me in consultation with Dr. W. L. Richardson.

A male infant (Case 322), healthy at birth, was suddenly attacked when it was six days old with general clonic convulsions, accompanied by a temperature of 40° C. (104° F.) in the first twelve hours, and afterwards to the time of its death by a temperature of 38.8° C. (102° F.). The attack followed a period of nervous excitement in the mother, who was nursing it, and who in other respects showed no abnormal symptoms. There were no symptoms of cerebro-spinal meningitis, such as retraction of the head or opisthotonos, and in the intervals between the convulsions, which occurred about every hour, the infant seemed perfectly well. It did not vomit, and did not have any abnormal symptoms connected with the eyes. The convulsions, which constituted the only symptom, continued, and on the second day of the attack the infant died suddenly.

The report of the autopsy, made by Dr. Whitney eighteen hours after death, was as follows:

Rigor mortis well marked. Lividity of the dependent parts of the body, and in small separated patches over the arms, legs, and chest.

The calvaria presented nothing abnormal. The inner surface of the dura was covered with opaque yellowish patches of lymph, especially marked over the base of the skull. The vessels of the pia mater were markedly injected, and its meshes were filled with an opaque greenish-yellow exudation. This exudation extended over the entire brain and into the spinal canal. Microscopic examination showed the presence of large micrococci, usually associated in pairs, two of which were sometimes united with a chain of four (pneumococcus).

The heart was normal, and its cavities were filled with dark fluid blood.

The lungs were not fully retracted, and were engorged with dark blood, which was so abundant as to suggest extravasation into the alveoli. The pleural surfaces were perfectly smooth.

The abdominal organs—spleen, liver, and kidneys—were markedly injected with blood, but were otherwise normal.

The stomach and intestines presented nothing abnormal.

The pathological diagnosis was, acute purulent cerebro-spinal meningitis, and general venous congestion.

**PROGNOSIS.**—The prognosis, where the child is young and the onset is violent, with high temperature and continuous convulsions, is very serious; but, even in the apparently fatal cases where coma has intervened, a change may take place and the child recover. The first two weeks are usually the critical periods, so far as the acute form of the disease is concerned. The disease varies in duration, sometimes lasting for only a few days, in other cases for a number of weeks; but in some cases it lasts for months, when it constitutes the chronic form, which is apt to prove fatal, both from exhaustion and from the development of more serious central nervous lesions.



**TREATMENT.**—The treatment of cerebro-spinal meningitis varies according to the severity of the symptoms. In most cases sedatives, such as the bromides, are indicated, and where the pain is severe opium in considerable doses is often needed. The ice-bag or Leiter's coil applied to the head, and absolute quiet in a darkened room, are important adjuvants to the treatment. In many cases the pulse becomes so weak and the prostration so marked that stimulants are needed until convalescence is established, when they can usually be replaced by tonics. In some cases the hyperæsthesia and general sensitiveness to noise, light, and motion in the room are so extreme and so characteristic that the attendants should be cautioned not to touch the child or the bed unnecessarily, and absolute quiet should be enforced in the room and throughout the house.

I have already told you that, as a rule, cerebro-spinal meningitis in children is a disease which is characterized by acute onset. This case, which I take from my notes, is illustrative of this fact:

A boy (Case 323), thirteen years old, had never had any especial diseases, but had been rather delicate for a number of months. He went to a Christmas party on December 25, and on returning from the party complained of the motion of the sleigh in which he rode home. On the following day, in the afternoon, he was found to be listless, to have his tongue coated but not dry, and to have a temperature of  $40.5^{\circ}$  C. ( $105^{\circ}$  F.) and a pulse of 140. He complained of tenderness and pain in the back of his neck; there was also tenderness in the abdomen. He appeared to be somewhat dull.

On the following day the temperature in the morning was  $39.4^{\circ}$  C. ( $103^{\circ}$  F.), and the pulse was 120. He was much more dull and apathetic than on the previous day, and in the afternoon became delirious. In the evening he had involuntary passages of urine and loose discharges from the bowels. His temperature was  $40^{\circ}$  C. ( $104^{\circ}$  F.).

On the following day his temperature was  $39.4^{\circ}$  C. ( $103^{\circ}$  F.), and the respirations varied from 40 to 80 and were regular. He was unconscious. *Subsultus tendinum* was present. There was retraction of the head. The pupils did not respond to light, but were equal in size. A *tache cérébrale* was present.

On the evening of the following day, four days from the onset of the disease, he died.

The autopsy made by Dr. Gannett showed the convex surface of the entire brain and cord to be covered with a thick exudation of pus, the spleen to be enlarged, and the case to be one of acute cerebro-spinal meningitis.

I have here in the wards a child (Case 324, page 697), two years old, who was brought to the hospital on the 21st of the month with the history that it had been showing symptoms of malaise for six weeks. Two weeks previous to entering the hospital it had a convulsion, and the indefinite and general symptoms had become more pronounced. There had been loss of appetite, with constipation; at times vomiting, slight cough, and a heightened temperature.

You see on examining the child the position which it assumes in bed. The head is retracted, and the muscles of the neck are rigid. The eyes are staring, but the pupils react to light. There is at times, though not now present, slight opisthotonos. On examining the child in front (I.), you see that the abdomen is retracted. Looking at it from behind (II.), you see that the occiput touches the back of the neck, and that the emaciation is extreme, so that the vertebræ and ribs have become quite prominent. The child is apparently unconscious, and does not notice objects which are brought before its eyes, although the eyes are open. It moans at times, and sometimes the legs are drawn up. No efflorescence has been detected anywhere on the skin. Although, as I have already told you, the onset of cerebro-spinal meningitis may be acute, yet in certain cases the prodromal symptoms are of a subacute character and somewhat prolonged, as has occurred in this case, which seems



to be one of this disease. It has been in the hospital for seven days, which would make the time since it was first noticed to be sick seven weeks. Since entering the hospital the temperature has varied from  $36.6^{\circ}$  to  $38^{\circ}$  C. ( $98^{\circ}$  to  $100.5^{\circ}$  F.). At intervals it has vomited and has apparently been unconscious. Sometimes it has cried out sharply, as though in pain. A *tache cérébrale* has been found at times, and the retraction of the head has been almost continuous.

The continuous retraction of the head, with at times opisthotonos and unconsciousness without the serious cerebral symptoms which after the fourth or fifth week would accompany an attack of tubercular meningitis, and the absence of any symptoms which point

## CASE 324.

## I.



## II.



Cerebro-spinal meningitis. Male, 2 years old.

towards disease of the thoracic or abdominal organs, lead me to make the provisional diagnosis in this case of cerebro-spinal meningitis. From what I have told you in a previous lecture in speaking of tubercular meningitis, especially of the recurrent form, an instance (Case 272) of which I showed to you at that time, you will understand that the diagnosis must be somewhat uncertain in a sporadic case of this kind until the disease has been under observation a still longer time.

The treatment of this case is simply the frequent administration of milk, with the addition of stimulants when indicated by the weakness of the pulse. The child has been in so apathetic a condition that the use of any drug has been unnecessary. Although at times it has cried out as if in severe pain, yet these attacks have not been sufficiently long to indicate their control by an opiate.

(Subsequent history.) During the following month the child remained very much the same as described above. The head was retracted at times, and the emaciation became extreme, the abdomen being very much sunken (boat-shaped). In the next two weeks the nourishment was taken more readily, the head was less retracted, and he began to notice objects around him, but he vomited once or twice nearly every day. The temperature at this time became normal.

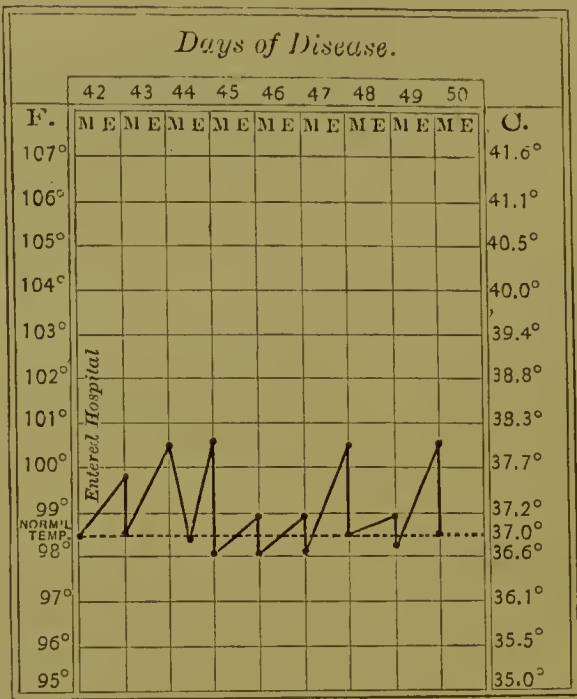
This chart (Chart 26, page 698) marks the temperature from the day when the child entered the hospital, in the sixth week of his illness, until the temperature became normal, nine days afterwards.

One month later, which was two months from the time when the child entered the

hospital, he was able to sit up without help. There was no retraction of the head, but the muscles of the neck were very rigid, and the head showed a tendency to fall back.

During the following month the child continued to improve slowly, increased in weight,

CHART 26.



recovered his appetite, and when seen one month later was found on physical examination to be in a normal condition. Here is a picture (III.) of the child, which was taken after an examination made by me which showed him to be in a normal condition in every respect.

CASE 324.

III.



Cerebro-spinal meningitis. Recovery after 4½ months.

This next child whom I have to show you is a girl (Case 325), eight years old, who apparently represents that form of cerebro-spinal meningitis which is designated chronic, and only a few cases of which have been reported.

The child entered the hospital two days ago. Her parents are said to have been healthy, and there is no evidence of tuberculosis or syphilis in the family. A brother whom I saw in consultation died of cerebro-spinal meningitis. With the exception of an attack of measles and of whooping-cough, the child has not had any other diseases. The present illness began four and a half months ago. The child had not been entirely well since the attack of pertussis which occurred one year ago.

The onset of this attack was sudden. She went to bed in fairly good condition, but woke up in the night delirious, screaming, and apparently not recognizing her parents. These symptoms continued until the following week. There were no convulsions. A week later vomiting occurred every two or three days. This was not dependent upon food, and it has occurred at intervals up to the present time. The bowels were constipated. There has been more or less opisthotonos from the beginning of the illness, and also in the beginning there was decided retraction of the head. The stiffness of the neck has gradually



diminished, but at times has been present since entering the hospital two days ago. Up to the present time the child is said to have had constantly a heightened temperature, varying from  $37.7^{\circ}$  to  $39.4^{\circ}$  C. ( $100^{\circ}$  to  $103^{\circ}$  F.), with a rapid pulse and quick respirations. Nothing abnormal has been found in the urine. There has been no efflorescence on the skin.

About four weeks ago the child was noticed to be blind. This has occurred suddenly. The child has had constant headache, and shortly after the beginning of the attack showed a loss of power of motion in both legs. At times there has been incontinence of feces and of urine. An examination of the urine showed it to have a specific gravity of 1015, to be normal in color, to have an acid reaction, and not to contain albumin or sugar. No evidence of syphilis was detected. She sometimes showed improvement in her general symptoms and became conscious, but she has not been able to sit up or to walk.

On physical examination you see (I.) that she is somewhat emaciated.

## CASE 325.

## I.



Chronic intermittent cerebro-spinal meningitis. Tache cérébrale showing on right thigh. Female, 8 years old.

There is extreme hyperæsthesia of the body and extremities. The slightest motion of the bed seems to cause discomfort and pain. An examination of the thoracic and abdominal organs shows that they are normal. The pulse is 80 and regular, the respirations are natural, the temperature is  $37.7^{\circ}$  C. ( $100^{\circ}$  F.). This morning she had an attack which was characterized by spasmodic contractions of all the muscles of the body, lasting for about thirty seconds. At this time there was no loss of consciousness, and the child screamed for some time afterwards as though in pain. During the attack the pulse grew feeble and intermittent, the respirations slow and superficial, and the extremities cold. Brandy was given subcutaneously, and reaction took place, so that she is now comparatively comfortable.

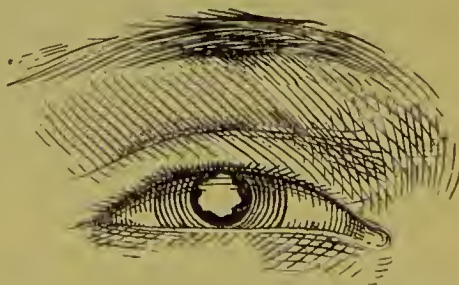
On examining the eyes (II.) you will see that although the pupils react and the retina is evidently sensitive to light, yet apparently she is blind.

You will notice in the middle of the eye a yellowish mass with an irregular border. Dr. Jack's report of the examination of the eyes is as follows:

"There is a very slight hyperæmia in the ciliary region. The iris seems slightly pushed forward, and its pupillary edge is a little uneven. A yellowish or yellowish-white reflex

## CASE 325.

## II.



Metastatic choroiditis occurring in cerebro-spinal meningitis.



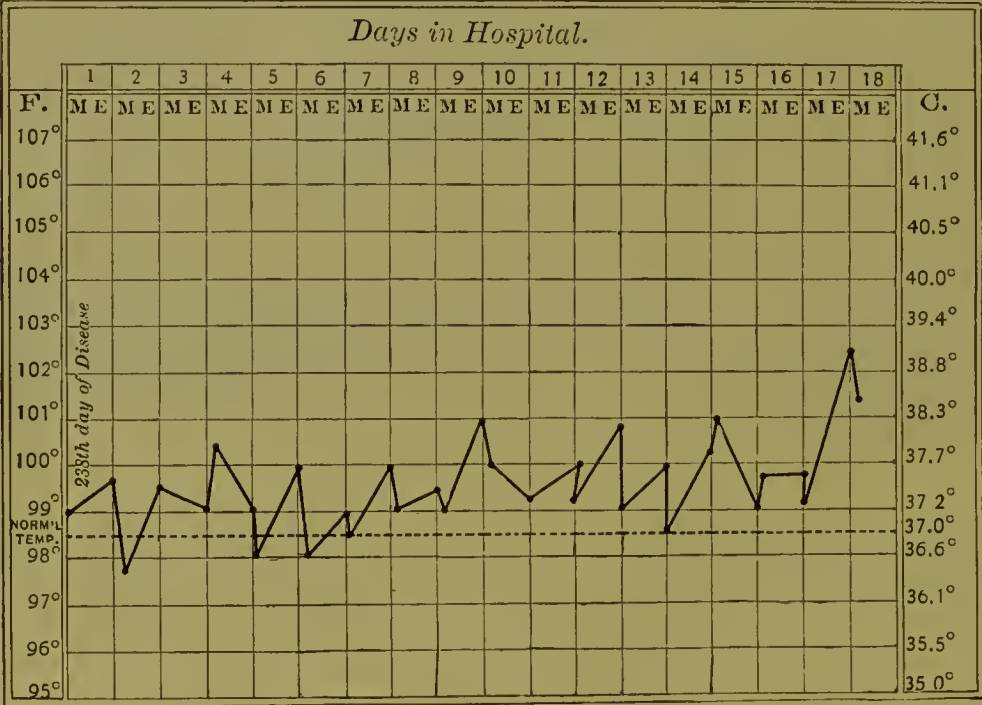
appears from the fundus of the eye even without the use of the ophthalmoscopic mirror, and it is easy to distinguish that this reflex does not come from the level of the lens, but that it is situated deeper. The tension of the eyeball is very much reduced, and there is very little tenderness on pressure."

These yellowish appearances in the pupils are sometimes called pus emboli. The disease is due to embolism, and is called metastatic choroiditis with an exudation of pus in the vitreous. It occurs quite frequently in cerebro-spinal meningitis. It is to be differentiated from glioma. Sometimes this yellowish mass fills the vitreous entirely, sometimes only in part. It may have blood-vessels on its surface.

You see on drawing the finger over the right thigh that a decided *tache cérébrale* is produced, which lasts from ten to fifteen minutes.

This is only the third case of this form of cerebro-spinal meningitis which has come under my observation. In both the other cases the children eventually died from a prolonged sickness of many months, during which they at times seemed to be recovering. Cases have been reported by others, as by Henoch of Berlin, to have recovered, so that in this especial case we are not able to give a more definite prognosis. At present there is no lesion which I have detected that would prevent the child from recovering, although she will always be blind. On the other hand, she may eventually die from exhaustion.

CHART 27.



Chronic cerebro-spinal meningitis.

(Subsequent history.) After the above report the child remained in about the same condition. At times she screamed as though in pain, but she took her nourishment fairly well. She had one slight convulsive attack, which involved mainly the upper extremities, the lower extremities being only slightly contracted. During this attack her thumbs were turned in, her fingers clinched over them, and her arms, which were usually extended at her sides, were flexed at the elbows. Her face showed no sign of spasm, and during the attack the radial pulse was full, soft, and regular. After a few seconds the muscles again became relaxed, and there was no further tendency to contraction. The usual position in which she lay during the following weeks was with the thighs slightly flexed and abducted and the legs flexed at the knee, with the heels almost touching each other. About two weeks after entering the hospital the right leg became flexed on the thigh to such an extent that the knee almost touched the chin and the heel rested on the vulva. Any attempt to extend the leg made the child cry out as though in pain, the left leg being naturally extended in bed. This condition of the right leg continued for several days and then disappeared.

Some days later a slight convulsive attack took place, which seemed to affect the right side more than the left.

This chart (Chart 27, page 700) shows the temperature during the eighteen days when the child was in the hospital. The pulse during this time varied from 68 to 100; the respirations sometimes varied from 34 to 52, but were usually about 28.

The fingers were flexed most of the time, and there was so much rigidity of the limbs that the reflexes could not be satisfactorily determined. The pus embolus in the right eye

## CASE 325.

## III.



## IV.



Chronic cerebro-spinal meningitis. Spastic condition of extremities 5½ months after onset of the disease.

seemed to be farther back from the plane of the iris than at the previous examination. The embolus of the left eye remained in about the same plane with the iris. The head was held rigid in any position in which it was placed, and she cried when it was moved. The pulse was 166, weak and compressible, the respirations were rapid, 42, the alæ nasi moved somewhat, and there was apparently a slight degree of dyspnoea. The temperature in the axilla was 38.6° C. (101.5° F.). There was slight cyanosis of the cheeks and lips, and an eruption



of milia on the chest, apparently arising from her continually perspiring day and night. She lay in a stupor all the time, except when she was moved, when she would cry out. She showed no signs of understanding anything that was said to her. Sometimes she would be seized with an attack of rapid breathing lasting several hours. The bowels had been constipated up to within two days, when diarrhœa occurred. There was incontinence of feces and urine, but no vomiting. During the last few days previous to this examination the teeth were kept closed, and had to be forced apart when she was fed. She was reported to have had one week previous to this examination a convulsion, in which the head was drawn back, the body and extremities were rigid, and the eyes rolled up. The child cried out sharply just before the convulsion. A physical examination made at this time showed nothing abnormal in the chest or abdomen.

## CASE 325.

## V.



Chronic cerebro-spinal meningitis. Spastic condition of hand 5½ months after onset of the disease.

Eighteen days after entering the hospital the child was taken to her home, so that the daily record could not be obtained.

An examination made two weeks after she left the hospital showed a spastic condition of the extremities and neck, as seen in these illustrations (Case 325, III. and IV., page 701).

When seen by Dr. Bullard at this time the child took no notice of her surroundings, and her eyes when opened had a vacant expression, due largely to the mental condition, although at this time she was undoubtedly blind. The extremities were much wasted, and were all in a condition of spastic rigidity. There was slight flexion of the thighs on the body and of the legs on the thighs, while the feet were extended in nearly a straight line with the legs.

The hand, as you see in this illustration (V.), is flexed almost at right angles to the wrist. The proximal phalanges of the fingers are hyperextended, while the other phalanges are flexed. The thumb is strongly adducted, and its distal phalanx is flexed.

This is a position of the hand frequently found in the later stages of spastic paralysis, and is due to the preponderant contraction of the flexors of the wrist and weakness of the interossei and lumbricales.

(The child gradually grew weaker, and died of exhaustion a few weeks later.)

I will also report to you another of these rare cases of chronic cerebro-spinal meningitis, which I saw in consultation with Dr. Townsend.

The child (Case 326), a boy, four and a half years old, had been previously well, with the exception of an attack of measles when he was one year old.

On May 9 he was suddenly attacked with vomiting, which continued at intervals for two days. From the beginning of the attack he complained of severe pain in the head and abdomen. On the second day of the attack there was much contraction of the head, and he was slightly delirious, although rational most of the time. The temperature was raised from the beginning of the attack. There were no convulsions. The bowels were not moved during the first week of the disease. When first seen by Dr. Townsend the pulse was 124 and regular, the temperature 38.8° C. (102° F.), and the respirations 20 and regular. There was slight opisthotonos. There were no contractions of the muscles of the limbs. The symptom of Kernig was present. There was no tenderness along the spine. The cutaneous sensibility was everywhere normal. There were no cutaneous efflorescences or ecchymoses. The pupils were regular and reacted normally to light. There was no strabismus or photophobia. Nothing abnormal was detected on physical examination. The patient was apparently in great pain, cried out a great deal, and moaned continually. The suffering during the next few days was so great that morphine in doses of 0.002 gramme ( $\frac{1}{30}$  grain) had to be given. This dose had to be increased so frequently that it was found that the child took 0.015 gramme ( $\frac{1}{4}$  grain) before relief was obtained.



Application of ice to the head and spine gave no relief, and for a number of days later it was found that there was needed to control the restlessness and pain 0.01 to 0.02 gramme ( $\frac{1}{8}$  to  $\frac{1}{4}$  grain) of morphine during the twenty-four hours.

On the twenty-fourth day of the disease the temperature, which had varied from  $37.7^{\circ}$  to  $39.4^{\circ}$  C. ( $100^{\circ}$  to  $103^{\circ}$  F.), became normal, remaining so until the forty-seventh day. During this time the head was only slightly retracted, and the child seemed free from pain, but remained in a very listless condition, not speaking, and taking but little nourishment or stimulants. He became emaciated, passed his urine and faeces involuntarily, and occasionally vomited. Nutritive enemata were not retained, but on the forty-first day of the disease peptonized milk was retained, and on the forty-sixth day he was able to take gruel, and at that time talked and laughed.

On the forty-seventh day of the disease a relapse occurred, the temperature rising to  $39.7^{\circ}$  C. ( $103.6^{\circ}$  F.). The head was rigidly drawn back, the eyes were staring, and the pain returned. The symptom of Kernig, which had never disappeared entirely, again became well marked. At this time I saw the child with Dr. Townsend. On the sixty-sixth day of the disease the convulsive movements of the left arm and leg, with turning in of the left eye, occurred. Several days previous to this relapse a number of sudamina appeared on the neck and trunk, and an evanescent erythematous eruption on the neck and face, lasting only a few hours. From the sixty-first to the sixty-sixth day of the disease his body was covered with a macular efflorescence, the macules varying in size. Eechymoses were at no time seen, and repeated examinations of the chest and abdomen showed nothing abnormal.

From the seventy-first day to the seventy-third day the temperature was again normal, the child took his food well, the neck was straight, and his general appearance was encouraging.

On the seventy-fourth day he again had convulsive movements, most marked on the left side of the body. The head was drawn back, and at noon the next day his temperature was  $39.4^{\circ}$  C. ( $103^{\circ}$  F.). The pulse, which during the entire illness ranged from 120 to 140 and had previously been regular, was now at times irregular and intermittent. The bowels were constipated at this time.

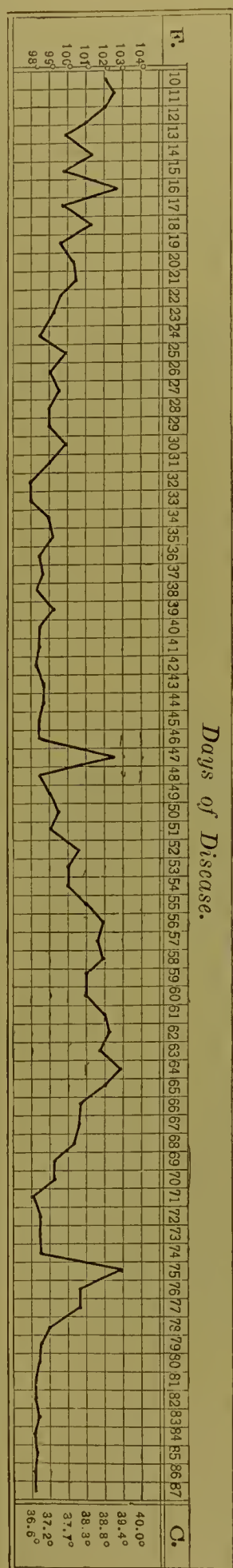
After this, although the temperature became normal, the child failed rapidly, and there was so much emaciation that the finger and thumb could easily encircle his thigh.

He died quietly on the eighty-seventh day from the time of the onset of the disease.

Through the kindness of Dr. Townsend I am enabled to show you his temperature chart (Chart 28) from the tenth day of the disease.

It was very difficult, indeed impossible, to give a prognosis in this case, as at times it seemed as though he would recover, and then the temperature would rise again, and the unfavorable symptoms would return.

Chronic intermittent cerebro-spinal meningitis.



## LECTURE XXXIV.

## PERIPHERAL NERVES.

NEURITIS.—PARALYSIS OF THE NEW-BORN.—NEURALGIA.

NEURITIS.—Neuritis is an inflammation of the peripheral nerves. It is accompanied by pain and tenderness in the affected regions, and in the more severe cases by paralysis and atrophy. I shall not dwell upon the cases of neuritis of a single nerve-trunk or its branches, which may be caused by traumatism, cold, or pressure, or may occur in the course of various diseases, but shall merely say a few words concerning a definite form of this disease, called *multiple neuritis*.

MULTIPLE NEURITIS.—In certain constitutional conditions a number of nerves in different parts of the body are affected with neuritis, and this constitutes the disease *multiple neuritis*.

ETIOLOGY.—Multiple neuritis usually occurs in the course of or subsequent to one of the infectious diseases. Of these diseases diphtheria is the most common, but it is said to follow scarlet fever and measles. A mild form sometimes occurs after typhoid fever. At times multiple neuritis is produced by drugs, such as lead, arsenic, or alcohol. It is not a common disease among children. The epidemic form of the disease has long been prevalent among the Japanese, and is known by the terms kakke and beriberi, but it is quite rare in this country, and I have never met with it in children.

PATHOLOGY.—The pathological condition in multiple neuritis is an interstitial or parenchymatous inflammation of the nerves. A few nerves may be affected, or the distribution may be general. The nerves of the special senses, however, are rarely affected, and the nerves of the head and face are not usually involved.

SYMPTOMS.—The onset of the disease may be acute or subacute. It may at the beginning present severe symptoms, such as extreme pain, tenderness over the nerve-trunks, and fever with an accompanying paralysis. On the other hand, the pain in the beginning may be very slight, and the first symptoms noticed may be a gradually increasing weakness of the limbs, while the tenderness may be found only when especially sought for. There may be hyperæsthesia, anæsthesia, numbness, and loss of muscular power. After the acute symptoms have passed away the faradic irritability is diminished: the action of the nerves to the galvanic current is diminished, and the reaction of degeneration is present. When the extensors of the leg are affected there is foot-drop, and when those of the forearm are affected there is wrist-drop. The course of the disease is apt to be a long one, and in the



later stages atrophy occurs, while the early hyperæsthesia may give place to a more or less marked anæsthesia, and numbness and various other paræsthesiæ may occur. In mild cases, where only pain and tenderness exist, the knee-jerks are not diminished, and may be even slightly increased, but in the more typical cases of the disease they are absent. Contractures and spasmodic conditions are absent, the paralysis being flaccid. The temperature is apt to be somewhat raised, and is decidedly so at the onset when the disease is acute.

**DIAGNOSIS.**—The diagnosis is to be made chiefly from poliomyelitis anterior, which may simulate multiple neuritis in certain cases; but in the former disease the absence of pain except during the first few days, with the more limited distribution of the paralysis, and the absence of tenderness, will serve to distinguish it from the latter.

**PROGNOSIS.**—The prognosis of multiple neuritis is favorable even where the disease begins with an acute onset accompanied by delirium and high fever, and, although the paralysis may last for many months, the cases usually recover.

**TREATMENT.**—The treatment is at first by absolute rest in bed, and later with electricity, massage, and strychnine.

Iodide of potassium is indicated in those cases which are caused by lead or arsenic.

In the subacute cases electricity and massage are indicated from the very beginning.

It is safer to wait until the pain and marked tenderness have disappeared before beginning the administration of strychnine.

This little girl (Case 327), eleven years old, represents a case of multiple neuritis produced by doses of 1 gramme (15 minims) of Fowler's solution given three times a day for some weeks during an attack of chorea.

The first symptoms which were noticed while she was taking the arsenic were that she vomited several times, but this was not supposed to have been caused by the arsenic, and the drug was therefore continued. It was next noticed that the child was unable to walk. Her limbs appeared to be very weak, and there was absence of knee-jerks and ankle-clonus. The sensation of the limbs was normal. A few days later she was found to have tender points over various parts of the legs. The legs then became atrophied. About a month later tender points developed in the arms, and she soon lost the power of using her arms, to such a degree that she had to be fed. At this time, although the arsenic had been omitted for several days, a large quantity of it was found by Professor Wood in the urine.

You see to-day that she has no remains of the choreic movements, but that she is rather stupid, and that there is tenderness on deep pressure over certain points in the calves of the legs. She has no headache and no other abnormal symptoms. She is being treated with the faradic current daily and with 0.18 gramme (3 grains) of iodide of potassium three times a day. Since this treatment was begun, three weeks ago, the

CASE 327.

Multiple neuritis. Female,  
11 years old.

power of grasping has returned, and the arms react somewhat better to the faradic current.

(Subsequent history.) One month later it was found that she could almost support herself without assistance. A little later she walked with crutches, and a month later she could walk without assistance, but with difficulty. The knee-jerks were still absent. She continued to improve, and finally after a number of months recovered entirely.

This boy (Case 328), who has been brought to the hospital this morning, is an interesting case of multiple neuritis.

He is nine years old. He was perfectly well until he was six years old, when he had an attack of measles. He was sick for two weeks, and then recovered apparently entirely. A week later it was noticed that he became fatigued on going up-stairs, and finally he lost the use of his limbs. For a year he walked with the help of a chair. There was simply loss of power of motion, but no pain. The appetite was not lost. He at times had slight headache. After the paralysis appeared it was noticed that the cervical glands swelled at times. The bowels were regular, and there was no trouble with the urine. The limbs were somewhat tender on pressure. His temperament was changed, so that he was rather fretful. Somewhat later he lost the use of his legs entirely, so that he had to be carried.

He then left his home and went to Florida, and after a few months recovered the use of his limbs entirely and became perfectly well. He returned to his home and went to school for a month. At the end of that time the symptoms of the previous attack began slowly to return, and he finally had to stop going to school.

On examination he is found to protrude the tongue straight. He has no symptoms referable to the head. There are red exanthematous patches on the elbows and knees. There is no especial atrophy of the legs, but there is a good deal of emaciation of the arms. The arms cannot be raised beyond the level of the shoulder. There is some pain in the shoulders when the arms are raised for him. There is tenderness on pressure of the shoulders. The flexion of the arms is good; the grasp of both hands is weak, but there is no loss of movement. The patellar reflexes are absent, and he walks with a peculiar tilt of the pelvis. He can stand well with his eyes shut. There is no disturbance of the kidneys and bladder, and no proof that the symptoms result from masturbation. No irritation is noticed about the prepuce, which, however, is tight. He cannot get up from a sitting posture or when lying down. He apparently has lost the power of pushing with his arms. The vertebral column is straight, and there is no apparent tenderness. He has never had diphtheria nor any of the eruptive diseases except measles.

The history of this patient and the examination lead me to eliminate hereditary ataxia and locomotor ataxia. The rapid improvement which took place in this instance when the child was taken away from his home for some months, and the recurrence of the symptoms within a month after his return, justify me in suspecting that the cause of the disease is a local one connected with the child's home. Of such toxic influences, that from lead is the most common and probable.

**PARALYSIS OF THE NEW-BORN.**—By paralysis of the new-born is meant that form of peripheral paralysis which occurs during the delivery, and which, as a rule, affects the face or one of the extremities. In this sense it is to be separated from injuries to the brain and spinal cord which are produced during the delivery,—in fact, from any paralysis of central origin which may occur in intra-uterine life, either before or at the time of delivery.

**ETIOLOGY.**—The cause of this form of peripheral paralysis is most often traction made upon the head of the child during delivery, thus producing a direct injury to the nerves, or dislocation or fracture of one of the bones, resulting in pressure on the nerves. Although this form of paralysis has



been known in a number of cases to result from pressure by the forceps during the delivery, yet it has also been met with after an apparently normal delivery, where the pressure did not seem to be especially severe or prolonged.

**PATHOLOGY.**—When the nerves of the face are affected, the resulting lesion is supposed to be from an injury of the facial nerve; and when the arm is affected, the lesion is supposed to be an injury of the brachial plexus or of the nerves in the lower part of the neck. When the limbs are affected, both arms have been known to be paralyzed; but, as a rule, the lesion is of one arm.

**SYMPTOMS.**—A paralysis of this form becomes apparent immediately after birth. This is a very important fact to remember, as in this way we can differentiate the disease from a paralysis resulting from poliomyelitis anterior, which is exceedingly rare in the early months of life, the youngest case on record being twelve days old. Where the face is affected, it is due, as a rule, to an injury of the seventh nerve, thus producing a peripheral facial paralysis. The peripheral form of facial paralysis is distinguished from the central in that in the former all three branches of the seventh nerve are apt to be affected, while in the latter form only the lower two branches are involved. In the peripheral form, therefore, the eye on the affected side cannot be closed entirely, causing the condition known as *lagophthalmia*, and there is inability to wrinkle the muscles of the forehead on the affected side. In facial paralysis of central origin the muscles of the forehead are not affected, and the ability to close the eye is but little decreased.

Where the paralysis affects an arm it hangs lifeless by the side, with the palm turned backward and the fingers often flexed. The fingers and forearm may be moved, but the movement of the upper arm to any extent is lost.

**DIAGNOSIS.**—This form of paralysis is to be diagnosticated from cerebral paralysis by the absence of increased reflex irritability and by the distribution of the paralysis. In the cerebral form all the muscles are affected; in the peripheral form, only individual muscles. It is doubtful whether paralyzes of spinal origin occur in the early days of life.

Cases of paralysis of the arm in the new-born should also be diagnosticated from surgical injuries represented by fractures, dislocations, and separation of the epiphyses. These are eliminated only by a careful examination of the head of the humerus on the affected side, showing the absence of crepitus, abnormal mobility, callus, or deformity.

**PROGNOSIS.**—The prognosis in cases where the face is affected is very good, as the paralysis in these instances lasts but a short time. We must, however, be somewhat guarded in the opinion which we give concerning them, as in some instances the paralysis does not disappear and the muscles of the face are left irreparably injured.

In regard to the paralysis of the arm, the prognosis is generally un-

favorable, especially if marked improvement does not soon occur, and ordinarily when improvement takes place it is very slow. Most of these cases never recover, and partial recovery should not be expected for a number of years. Shortening of the arm is marked in the later history of the severer cases.

#### CASE 329.



Peripheral paralysis of the new-born. Paralysis of right side of face. Forceps delivery. Infant, 2 hours old.

We can therefore state that peripheral paralysis of the new-born when it affects a limb is much more serious in its prognosis for complete recovery than when it affects the face.

**TREATMENT.**—Electricity and massage continuously applied for a number of years is a very important part of the treatment of these cases, and obviates the atrophy of the muscles from disuse, which must necessarily take place to a greater or less extent.

Here is an infant (Case 329), two hours old, who has a peripheral paralysis of the right side of the face, caused by pressure of the forceps on the seventh nerve.

In this case the closure which you notice of the right eye is produced by the swelling of the face and eyelid. You see that the entire right side of the face is affected.

I have here another infant (Case 330), one year old, who presents the condition of peripheral paralysis of the right side of the face.

#### CASE 330.



Peripheral paralysis of the new-born. Paralysis of right side of face. Infant crying. Male, 1 year old.

When the infant cries you see that the lines on the right or paralyzed side of the face are somewhat obliterated, and that the right eye cannot be closed (lagophthalmia). The lines of the left or non-paralyzed side of the face, on the contrary, are deepened, and the left eye can be closed. The mouth is drawn to the left.

The prognosis of this case is bad for complete recovery, and treatment of any kind will probably be of no avail, owing to the length of time for which the lesion of the seventh nerve has existed.



This little boy (Case 331) is two years old. He was healthy at birth, but the labor was instrumental. When he was three days old it was found that his left arm was swollen. He was first seen at the hospital when he was seven weeks old. At that time he was able to move his fingers and wrists, but held his arm with the elbow straight to the side and the hand pronated. He is now, as you see, able to make slight movements of flexion of the

CASE 331.



Peripheral paralysis of the new-born. Paralysis of left arm. Male, 2 years old.

elbow and slight contractions of the deltoid. Under the use of electricity he has been showing gradual improvement. He can grasp objects fairly well with his left hand, and can flex the elbow completely, and raise his hand and forearm as far as the nipple. You see he can raise his right arm with ease to his head, but cannot raise the left hand farther than the lower part of the chest.

It is evidently a case of paralysis of peripheral origin caused by trauma.

The prognosis in this class of cases is often grave for complete recovery, but, as you see, considerable improvement has taken place in this child.

**NEURALGIA.**—In contradistinction to the affection of the nerves which I have just described as neuritis is a functional affection of the sensory fibres of the peripheral nerves, represented by pain and called neuralgia.

Neuralgia is so rare in infancy and early childhood that I shall not do

more than refer to it. When neuralgia occurs it may affect very different localities, and may be represented by intercostal neuralgia or the various milder forms of flitting pains in different parts of the body which so commonly occur in children.

I have found in most cases of neuralgia that temporary relief from the pain can be obtained by the use of phenacetine, and I have never seen any contra-indications to using this drug. It can be given in doses of 0.06 gramme (1 grain) for every year of the child's life up to 0.6 gramme (10 grains). I am in the habit of guarding against any possible bad effects by giving the phenacetine in a little brandy-and-water.



## LECTURE XXXV.

## II. NERVOUS DISEASES PRESUMABLY ORGANIC.

## CHOREA.—EPILEPSY.—INSANITY.

IN speaking of the next class of nervous diseases, which I have called "presumably organic," it may perhaps be well to explain why I have made use of this term. It is because we cannot help feeling that in true chorea or true epilepsy there must be some organic lesion, and that it merely remains for future investigation to show what the lesion is. When this lesion has been determined we can relegate the disease to the organic class, or possibly it may be decided that it belongs to the functional diseases. Of course there can be but a slight pathological distinction between these diseases and those which I have called functional, but their chronic course and their serious nature ally them clinically so much more closely to the diseases of known organic origin than to the indefinite functional class that, for simplicity in teaching, I have decided to separate them from the latter.

**CHOREA.**—Chorea is a disease characterized by irregular and involuntary muscular movements without loss of consciousness, and affecting the muscles of volition.

The disease is rare in infancy, but may occur in the early months of life. It seldom begins after puberty. It is most apt to begin and is most marked in its symptoms during the period of the second dentition,—that is, during the period of active growth, from six years to puberty. The greatest number of cases is found among the female sex and among those who do not receive sufficiently nutritious food. It will be well for you to understand clearly that a sharp distinction should be made between the disease chorea, with its characteristic choreiform symptoms, and the same choreiform symptoms resulting from various diseases, sometimes represented by central nervous lesions, sometimes by purely reflex causes. It will save you much useless reading of the literature of chorea and much profitless discussion as to its etiology and pathology if you will bear this distinction in mind. Eliminating those forms of chorea which are due to gross lesions of the nervous system, such as the post-hemiplegic and congenital forms, we can at once very materially reduce the cases of true chorea. In like manner we should separate from true chorea those cases of peripheral irritation in which the partial choreiform symptoms are evidently reflex and can be cured by removal of the cause. Examples of these reflex choreiform symptoms are the facial chorea from naso-pharyngeal irritation and the partial choreiform movements occasionally arising from errors of refraction and ocular insufficiency. The consideration of these anomalous forms of

chorea belongs with the diseases in which they occur, and they should be spoken of in connection with the other symptoms of these diseases.

**ETIOLOGY.**—Chorea can be precipitated by other diseases, such as measles, though this, in my experience, rarely occurs except among the poorly cared-for. A certain number of cases have so directly followed intense fright that we must acknowledge acute mental conditions as a cause. The disease which is most frequently associated with chorea is rheumatism. The percentage of cases, however, in which this association takes place is difficult to determine. This difficulty arises from the want of uniformity in the reported cases of different observers, due to their different ideas as to what constitutes rheumatism. If only the cases of acute articular rheumatism are to be classified under rheumatism, very few cases of associated chorea will be spoken of; while if all the flitting aches and pains of childhood are considered to be rheumatism, the number of choreic cases caused by rheumatism rises to fifty per cent., or possibly more. The truth will in the future probably be found to lie in some intermediate number, for that in certain cases a close connection exists between chorea and rheumatism is very evident. The difficulty becomes still greater when we examine the relationship between chorea and endocarditis. Of course where there is a rheumatic element in the case we should expect a cardiac lesion to arise, and to be dependent on the rheumatism. In certain cases, however, we find chorea with endocarditis entirely irrespective of rheumatism. This occurs to such an extent that in our cases of chorea we should watch for cardiac lesions just as carefully as in our rheumatic cases. Heart-murmurs of a hæmic nature may occur in chorea as in any other disease of a debilitating nature. They should, however, always be looked upon seriously, as possibly indicating an insidious form of organic endocarditis, which, instead of being evanescent and passing off entirely with the recovery of the chorea, may either seriously disable the heart or lead to a fatal issue. A special microbic cause for chorea, as for rheumatism, must be thought of, but as yet has not been proved to exist. An hereditary tendency to nervous explosions of a choreic type has long held a prominent place in the etiology of chorea. In my experience, however, it is not very common, unless the children are poorly nourished, badly cared for, or exposed to nervous excitement during their school life.

Overtaxing of the central nervous system during the school year has so often been shown to result in an attack of chorea in the spring and in a recurrence in the autumn on returning to school, that it should be recognized in considering the etiology of the disease. Strain of the ocular muscles has been considered an exciting cause of chorea.

**PATHOLOGY.**—There are a large number of cases of chorea in which the disease is found to have no apparent pathological lesion. Its symptoms, however, show us that the morbid process is located in some part of the central nervous system. The lesion, however produced and whatever it is, is represented by a profound excitement of the motor centres, presumably



due to their inanition, and is accompanied by a temporary inability of these centres to recover themselves. Many lesions have been described as occurring in chorea, but in the pure cases (Sydenham's chorea) which I have just described, and which really represent the disease, there is no lesion which with our present knowledge we can say is characteristic.

**SYMPTOMS.**—Chorea may be in its distribution general or partial; in its course acute, subacute, or chronic. In many cases the disease is exceedingly mild in its symptoms and of a benign type; in others it assumes a severity which seems to threaten life. I shall presently show you examples of both types of the disease. The beginning, though at times sudden, as from fright, is, as a rule, gradual, at first a few muscles only being affected. The child becomes fretful and impatient, and we must carefully differentiate these symptoms from those resulting from bad temper, for which they are apt to be mistaken by the family. The clinical picture of the disease is a jerky, irregular, involuntary contraction and relaxation of the muscles, apt to begin in the fingers, hands, and face. There is an irregular, uncertain action of the part affected, and efforts of the will only partly control the movements. As the disease progresses, the voluntary control of the muscles diminishes more and more, and at times disappears entirely.

The movements ordinarily cease during sleep, but in severe cases they continue during and even interfere with it. At times the child is unable to walk, on account of weakness. The speech may become slow and indistinct, from the affection of the muscles of the tongue and of the larynx, and even mastication and deglutition may become difficult. In very severe cases the difficulty in speech may be enhanced by the mental condition, which may become impaired, and which is represented by dulness and apathy. The tendon reflexes are apt to be lessened in severe cases. The muscles grow weak and soft, and there is considerable emaciation. There is usually loss of appetite, and the bowels are often constipated. The urine and its urica have been found to be increased during the course of the disease. The dynamometer usually shows impaired muscular power. In certain cases the muscles of the extremities on one side of the body are principally or alone affected (hemichorea). These cases do not differ from the ordinary bilateral cases in any way except in this respect.

In very severe cases there may be involuntary evacuations of the feces and of the urine. The disease is distinct from epilepsy, and there is little danger of the patient becoming epileptic unless the disease happens to develop in an individual who is predisposed to that condition.

**PROGNOSIS.**—Chorea is very apt to show relapses and to recur every year for some years. Though often obstinate in the persistency of its symptoms, yet it may be said to be self-limited, and, as a rule, to recover, provided no complications, such as from cardiac disease, arise. The time which elapses before complete recovery is very variable, but well-marked cases usually extend over a period of three or four months. Although, as you will notice, in speaking of the prognosis of chorea I consider it,



as a rule, a benign disease, yet we must always look upon it as a serious disturbance until we are sure that we are dealing with the usual mild form of the affection. As an illustration of how careful we should be to give a guarded prognosis in the early stages of acute chorea, I shall mention the following case :

A girl (Case 332), nine years old (Cook and Beale), began to have choreic movements, which constantly became worse. Delirium developed, with a slight fever, a rapid and feeble pulse, and a quick and interrupted respiration. Death suddenly occurred one hundred and thirty hours after the onset of the disease. The autopsy revealed extreme anæmia of the pons and medulla, but no other changes of note in other parts of the body.

We must allow that even uncomplicated chorea is a varying disease as to the severity of its symptoms and their persistence for a longer or a shorter time. We also know that there is a marked tendency to relapse, and that the number of relapses varies to a great degree. The length of the attack and the response to treatment may differ much. Bearing these facts in mind, you will comprehend the rapidity with which certain individuals are attacked or the quickness with which they recover. You will meet with some cases which recover rapidly under only hygienic treatment, and with others which are apparently unaffected by any drug whatever. Where heart-murmurs, evidently representing organic disease, appear, you will often find cause for wonder in the comparatively slight discomfort which the cardiac lesions entail. At times, again, you will be surprised at the rapidly fatal course of some cases complicated by cardiac disease, and at their uncontrollability by any treatment whatever.

**TREATMENT.**—The disease is variable in its duration whether treated by drugs or not. There cannot be said to be any specific treatment with drugs for chorea, but of the many drugs that have been used in this disease arsenic has, in my experience, been the most beneficial. Arsenic should, however, be used with care, and on the appearance of any evidence of the physiological action of the drug, such as nausea or oedema of the eyelids, it should at once be discontinued. It should not, as a rule, be given in very large doses, as cases have occurred in which it has produced a multiple neuritis of many months' duration. Where any special cause can be found for the attack, such as rheumatism, appropriate treatment directed to that cause should be employed. It is manifest, however, in the uncomplicated cases that our main reliance must be placed on hygiene and food. Fresh air, nutritious food, tonics to control the anæmia and general prostration, kindness, seclusion to secure mental quiet, stimulants if there is much resulting weakness, and the bromides for insomnia and over-excitement, are the means which I have found most valuable in managing this disease. I have seen well-marked cases get well in from sixty to seventy days where good food and a small amount of stimulant constituted the entire treatment.

If the attack is very severe, skilled nursing is a very important adjunct in the treatment. The child should be protected from harming itself by

means of the padded bed, and light but well-padded splints to control the movements during sleep are indicated occasionally.

I have a number of cases of chorea to show you. It is one of the most common diseases that are seen in the hospital.

Here is a little girl (Case 333), six years old, who represents one of the milder forms of chorea. There is no history of nervous or cardiac disease or rheumatism in the family, and the child herself has never been sick before.

Three weeks ago she complained of pain in her left hand and arm, and later the muscles of the arm began to twitch. Soon after, the whole body was affected in the same way. Somewhat later it was found that the child could not talk plainly, and it was with some difficulty that she could feed herself. She seemed nervous and peevish, and showed constant irregular incoördinate movements, chiefly of the face, mouth, and upper extremities. The legs were slightly affected, and sometimes the muscles of the trunk also. There has been no paralysis of the muscles. The eyes have been normal in their reaction.

Since entering the hospital she has been treated chiefly without drugs, and especial attention has been paid to giving her a nourishing diet, baths, gentle massage, and rest in bed in a quiet corner of the ward.

On entering the hospital, three weeks ago, a physical examination showed nothing abnormal in connection with the heart or other organs. An examination of the urine showed it to be normal.

You see to-day that she is looking very well, and that the incoördinate movements have ceased entirely. Marked improvement was shown after she had been in the hospital for two weeks, and for the last few days, about the forty-second day from the onset of the disease, I have considered her cured and ready to return to her home.

Here is a little girl (Case 334, page 716), eight years old, whose symptoms are so characteristic that we can at once make a diagnosis of chorea.

I have not been able to ascertain anything concerning the history of this child, except that she has been subject to attacks of this nature for some time. There is no history of rheumatism, nor of any other disease. The child seems to be physically well and strong, and on examination nothing abnormal is found in any of the organs, with the exception of a slight systolic souffle heard distinctly at the apex of the heart and transmitted through the axilla into the posterior scapular region. The area of cardiac dulness is not increased. The patellar reflexes are increased. There are marked choreic movements of the hands, legs, and head. On inspection you see that the limbs are flexed and extended, with irregular incoördinate movements, and that there are from time to time the same muscular contractions in the face. She shows a certain amount of mental disturbance, characterized sometimes by peevishness and sometimes by slyness; she is dull rather than bright.

She represents the disease chorea of a moderately severe type, which from its constant recurrence and chronic course will probably prove to be very intractable. The prognosis as to her mental condition is especially serious, as the probability is that the mental impairment will increase rather than decrease.

She has been treated with a number of drugs, such as arsenic, quinine, iron, and others, none of which seem to be of any benefit.

In regard to the mitral systolic souffle to which I have just referred, it is possible that it is wholly a functional manifestation. You must always bear in mind, however, that there is great liability in cases of chorea of organic cardiac disease developing, and that until all signs of cardiac disturbance have disappeared a very guarded prognosis should be given as to whether the cardiac disturbance is of functional or of organic origin.

In a case of this kind, instead of the gradual diminution of the murmur, which would seem likely to occur, judging from the very slight evidence of cardiac disturbance present, especially as the murmur could be well accounted for by the choreic functional disturbance, it is possible that an endocarditis with valvular disease may exist and later produce more serious symptoms.



I shall now show you this little girl (Case 335), thirteen years old, who represents one of the milder forms of recurrent chorea.

She had a number of diseases preceding her first attack of chorea. When she was two years old she had an attack of diphtheria, when five years old one of measles, when seven years old one of scarlet fever, and when eight years old one of rheumatism.

When nine years old she had her first attack of chorea, which occurred in the spring of the year and lasted for a number of months. This was followed in the spring of the next year by a second attack. In the spring of the following year she had a third attack of the

CASE 334.



Chorea. Female, 8 years old.

CASE 335.



Recurrent chorea, fifth attack. Female, 13 years old.

disease; at this time the incoördinate movements were not so marked as in the previous attacks, but the debility was greater. When she was examined during this attack, it was found that the heart, although weak and somewhat irregular, presented no evidence of murmurs. The pulse was 84, the temperature was normal, and there were no signs of any other disease. She was treated at the hospital, and recovered in a few months. In the spring of the next year she had a fourth attack of chorea. At that time nothing abnormal beyond the choreic movements was noticed. She was treated with from 0.18 to 0.36 gramme (3 to 6 minims) of Fowler's solution three times a day, and in a few months left the hospital apparently well.

In the spring of the present year, one year from the beginning of the fourth attack, she entered the hospital with a fifth attack, for which she is now being treated. In this attack, after using Fowler's solution for a few weeks and not obtaining any especial benefit, I have given her iron and nux vomica. She is gradually improving under this treatment,



and, as you see, has a fairly good color, has a good appetite, and seems quite strong. There are some remains of the incoördinate movements, which especially appear when she is embarrassed by the observation of the people who are around her. You see she now has a slight twitching of the face and hands, and occasionally the hands, and especially the thumbs, are drawn inward with an arrhythmical movement.

The prognosis in this case is good. Although she has had five attacks of chorea, no organic lesion of the heart, nor any other abnormal condition, has resulted from them, and she will probably recover entirely, and will not continue to have attacks of the disease when she is a little older.

This little boy (Case 336), eight years old, is a case of chorea which I wish merely to show you as one in which the treatment by quinine was found to be followed by an increase in the choreic movements and to be entirely without benefit.

The child has always been of a nervous temperament and very studious, and has grown rather more rapidly than other children of the same age. He was taken sick one month ago, and has been in the hospital two weeks.

On entering the hospital he had the usual symptoms of chorea, incoördination on using his muscles, and difficulty with his speech. His mind was clear, and there was nothing abnormal detected about him except a cardiac murmur, apparently hæmic, and considerable general weakness, so that he walked with difficulty. He was at once treated with absolute rest and quiet in a room separate from the rest of the patients in the hospital, and small doses of iron were administered. His general condition improved gradually but slowly during this week, when it was thought advisable to endeavor to hasten his recovery by giving him quinine. Sulphate of quinine was administered in doses of 0.12 gramme (2 grains) three times a day, with orders to have it gradually increased to 1.2 grammes (20 grains) in the twenty-four hours. This treatment was continued during the last week until the amount of quinine taken in the twenty-four hours amounted to 1 gramme (15 grains).

Under this treatment he has grown steadily worse. The disturbance in speech has greatly increased, and he has lost the power of using his arms and legs. His mind is clear. There is no vomiting, but he has a certain amount of tinnitus aurium and a slight headache. As the deglutition is also beginning to be affected, I have considered it wise to omit the quinine.

(Subsequent history.) After the quinine had been omitted for twenty-four hours the child's general condition was decidedly improved. The treatment with iron was renewed, and he gradually recovered, leaving the hospital one month later in apparently good health; the cardiac murmur had disappeared, there were no incoördinate movements, and he could speak and use his arms and legs normally.

This next boy (Case 337), nine years old, has been subject to attacks of chorea for nearly four years. The attacks usually come on in the spring with considerable severity, and continue for nearly six months, gradually diminishing in intensity until the symptoms are scarcely noticeable. The child has a history of rheumatism, not, however, of a high grade. The attack from which he is now suffering began four months ago, and has been a quite severe one, so that he has been unable to control the movements of his hands and face during the day; they are, however, quiet at night.

On entering the hospital a physical examination showed a marked systolic murmur, heard most distinctly at the apex and transmitted to the axilla. The area of cardiac dulness was not especially enlarged. The urine was normal, and nothing abnormal was detected about the child.

He was at first treated with Fowler's solution, 0.12 gramme (2 minims), three times a day. After four days the choreic movements became less marked and the cardiac souffle less distinct. Two days later, however, the Fowler's solution had to be omitted, as it caused nausea and vomiting. At this time there was a double souffle, heard most distinctly over the left third interspace, close to the sternum. A few days later Fowler's solution was renewed, but, as it caused gastric disturbance, again had to be omitted, and it was found that it could not be given for more than two days at a time without causing puffiness of the face.

A month later the choreic movements had decidedly lessened and the cardiac murmurs disappeared.

To-day, two months from the time when he entered the hospital, he appears to be free from the disease. His muscular movements are natural, though his legs are slightly weak; he has a good appetite, and there are no abnormal symptoms connected with the heart.

I show you this case as representing one of recurrent chorea in which arsenic is not tolerated, and in which the indications for treatment are chiefly rest, good food, bathing, and massage in the beginning, followed later by the administration of some mild form of iron, such as the tartrate of iron and potassium. The cardiac disturbance in this case was in all probability functional rather than organic, as not only were the cardiac murmurs most distinct when the child was weak and anæmic, gradually growing less as he grew stronger and the anæmia disappeared, but also on the most careful physical examination I can now find no evidence of organic disease. You can therefore consider it a case of recurrent chorea with accompanying functional cardiac disturbance.

This little girl (Case 338), four and one-half years old, in this bed in a quiet corner of the ward, is an exaggerated case of chorea.

Until this attack she had been a healthy, bright, strong child. She had an attack of pertussis when she was two and one-half years old, and one of measles when she was three and one-half years old. She has never had rheumatism. She began to have choreic movements of a rather subacute type one year previous to this attack for which she has been brought to the hospital. The onset of this second attack was while she was going to school, and when she was in fairly good health. The muscles of the mouth and face were first affected, and she seemed to get very much excited when at school, without any apparent cause. The symptoms rapidly increased in severity, and she was brought to the hospital a few days ago.

I shall not give you the details of this case, as they differ very little from those which I have already described to you. I show her to you as representing one of the exaggerated forms of chorea.

The symptoms have increased since entering the hospital, and the muscular movements are so prominent, even at times occurring when she is asleep, that she has to be kept in a padded bed. She is apparently unconscious. She sleeps with the greatest difficulty, and she has been unable to speak since she entered the hospital. At times the head has been slightly retracted, but there has not seemed to be any especial rigidity of the muscles of the neck. The area of cardiac dulness is not enlarged. There is a slight cardiac murmur, heard most distinctly at the base of the heart. She is being treated with stimulants and as much milk as it is possible to make her swallow, but at present she is taking only about 473.11 c.c. (1 pint) in the twenty-four hours.

(Subsequent history.) The choreic symptoms lasted for some weeks, but gradually grew less violent, and the child finally recovered entirely.

An examination of the heart two years later showed that organic disease was present, as indicated by the enlargement of the cardiac area of dulness, a mitral systolic murmur at the apex, and an accentuated second pulmonic sound. At the time of this examination the child was found to be weak and delicate, and was evidently suffering from the effects of organic cardiac disease.)

In connection with the previous case I shall report to you a case which I saw in consultation with Dr. Boardman.

A boy (Case 339), ten years old, had always been delicate, but had had no special disease, such as rheumatism, until six weeks previous to the time when I saw him, when he was said to have had an attack of epidemic influenza. He recovered completely from the disease in ten days, and seemed as well as ever. Three or four days later he began to show symptoms of chorea. These symptoms gradually increased in severity, and finally were continuous, except when he was asleep. After he had had the chorea for one week he was unable to articulate, and began to have trouble with deglutition. He soon lost the power of controlling his limbs, grew very weak, and was confined to his bed. There was considerable



insomnia. In the second week of the attack the choreic movements became so violent as to endanger his falling out of bed. The temperature up to the time when I saw him, in the fourth week of the attack, was normal. The pulse varied from 140 to 150, and the respirations from 35 to 40.

When I saw him, at the end of the fourth week from the beginning of the chorea, his mind was perfectly clear; he had a little pain in the hands and shoulders, apparently from the continual movements. He was unable to articulate clearly. There was difficulty in swallowing, and he was considerably emaciated. Nothing abnormal was found in the lungs. The heart was beating tumultuously. The area of cardiac dulness was very slightly enlarged, but there were no cardiac murmurs.

The case was apparently one of primary acute chorea without complications. Although in many of these severe cases of chorea no evidence of cardiac disease can be obtained on physical examination beyond a slight dilatation of the left ventricle, yet some disease of the endocardium or valves may often be found at the autopsy. In these cases, however, the temperature is, as a rule, raised. In this case the continuous normal temperature and the absence of any signs of cardiac disease beyond a slight dilatation from the apparent weakness of the ventricular muscles seemed to indicate that it was a case of simple chorea without disease of any of the organs.

Although the child was carefully nursed and remedies of various kinds were employed to strengthen the action of the heart and to support his general strength, he failed rapidly, and died of exhaustion a few days after I saw him.

I will now show you this little girl (Case 340), eight years old, whom I have had placed in a warm room so that she can be examined naked without harm.

#### CASE 340.

##### I.



Rheumatic arthritis. Endocarditis. Cardiac enlargement. Chorea. Female, 8 years old.

The history given to me when the child entered the hospital was that the mother had been subject to attacks of rheumatism. This child has not had any especial diseases, with the exception of an attack of measles when she was three or four years old, until she had an attack of rheumatic arthritis six months ago. At that time she was confined to bed with fever, and with pain, tenderness, and swelling in all her joints, especially of the knees and fingers. Although she recovered from the acute symptoms of the rheumatism, she has since then never been able to use her arms and hands, nor has she been able to walk much. I have no record of the condition of her heart during the attack of rheumatism, but so far as I can ascertain there was no evidence of cardiac disease prior to the rheumatism. During the course of the rheumatism there were no other especial symptoms noticed, except that her disposition was evidently much changed and she became peevish and fretful.

One week before entering the hospital she began to have choreic movements. They were moderate in degree, but incessant. A few days later the incoördination of the muscles was also noticed when she endeavored to speak or to swallow. There were continual



choreic movements of the eyes, face, and fingers, and, although seemingly she could understand what was said, she was unable to speak clearly. She has been in the hospital ten days, and is, as you see, much emaciated. You will notice the incoördinate movements of all the muscles of the face, eyes, head, neck, body, and extremities. The peculiar look which occurs in these cases, and which can be expressed by embarrassment, is clearly shown here. The child feels that she cannot control her muscular movements, and cannot even fix her gaze on any object steadily. Although the case is a severe one, the mind is not affected beyond a slight degree of hebetude. On physical examination I find that the lungs are normal. On examining the cardiac region you notice that the impulse of the heart is outside of the mammary line and in about the sixth interspace. On palpation the contractions of the heart are found to be of an irritable nature, clearly felt, but not so strong as normal. At times there is a feeling as though the ventricular contractions were hesitating, and they are of an irregular form, which suggests that the incoördination of the other muscles is participated in by those of the heart. On percussion there is normal resonance to the right of the sternum and under its upper part as far as the third right interspace. There is dulness under the sternum, beginning at the second left interspace, extending across to the third right interspace, and involving the lower part of the sternum. I have marked the area of cardiac dulness in black. It extends upward to the left of the sternum as far as the second rib, then to the left and downward outside of the mammary line until it reaches the impulse of the heart in the sixth left interspace. On auscultation a murmur is heard most distinctly with the first sound at the apex of the heart, and is transmitted to the axilla and to both sides of the back. This murmur is transmitted to the base, but gradually lessens as the area of the large vessels at the base of the heart is reached. Nothing else abnormal is detected on a further physical examination of other parts.

This is evidently a case where during the course of a rheumatic attack an endocarditis in all probability developed. This endocarditis has been followed by enlargement, mostly represented by dilatation of the left ventricle. During the course of the rheumatism and of the cardiac complication the chorea has developed.

The prognosis in a case like this must be very guarded. In some instances the disease, or rather combination of diseases, grows rapidly worse, and the child dies seemingly from exhaustion. In cases of a milder form the child gradually recovers from its chorea and from its rheumatism, but is left with an organic disease of the heart from which it never recovers. The cardiac disease, however, can in most cases be much benefited by careful treatment, especially by rest in bed. In these cases the dilatation grows decidedly less, while the heart becomes stronger, and, as the chorea passes away, shows a normal area of dulness.

During the course of a case of this kind we must at any time expect in place of gradual improvement a decided increase in the severity of the symptoms. The valvular lesion of the heart may become much more extensive, assuming the ulcerative form which is usually so fatal. The pericardium may become affected, and broncho-pneumonia may occur as a complication. I shall therefore have to tell the parents of this child that she is in an extremely critical condition, and that for some days or weeks it will be impossible to say whether she will live or not.

The treatment of the case is with milk and stimulants. It is impossible for her to take solid food, and the milk is with the greatest difficulty introduced into her mouth. I will now have the nurse feed her (II., page 721), so that you can see how almost impossible it is for her to take the milk in her mouth or to swallow it, although she evidently is desirous of doing so.

I have tried various methods of administering the milk and stimulants in this case, but have found that the jaws close so spasmodically whenever a spoon is introduced between the teeth that the milk is usually spilled before she receives it. The method which I have found to be most successful is by this feeding-cup with a rubber nipple fitted to the neck of the cup. The rubber nipple is perforated with a large hole. The soft rubber does not incite the choreic movements of the jaw to the degree that anything hard would do. You see that by a little effort of sucking she takes the milk fairly well, though the difficulty in swallowing continues. The amount of milk which we endeavor to give her in

the twenty-four hours, and which I think is sufficient to support her strength until the acute stage of the disease shall have passed by, is 1419.33 c.c. (3 pints). She is also taking about 60 c.c. (2 ounces) of port wine in the twenty-four hours.

(Subsequent history.) The child remained in about the same condition for the next three or four days, when the temperature rose to 38.8° C. (102° F.), and on the following day to 40° C. (104° F.), and she complained of pain in the precordial region. On examination, in addition to the cardiac murmurs a friction-sound was heard all over the cardiac area, but especially in the neighborhood of the left nipple. The area of percussion dulness remained the same, and did not extend to the right of the sternum. The child moaned considerably during the day, and was very restless. The choreiform movements becoming more

## CASE 340.

## II.



Administration of milk in a severe case of chorea.

exaggerated, the port wine was increased in amount and 3.75 c.c. (1 drachm) of infusion of digitalis was given once every eight hours. Under this treatment the pulse grew a little stronger and the child's restlessness became less. The temperature also fell to 38.6° C. (101.5° F.), and on the following day to 38° C. (100.5° F.). The pain continued during the next few days, and there was a certain amount of diarrhoea. The stimulant was increased to 120 c.c. (4 ounces) in the twenty-four hours. The child then became less restless, took more nourishment, and slept better. The diarrhoea ceased on the seventeenth day from the time when she entered the hospital, and at this time she began to swallow better and to speak more distinctly. During the next few days her general condition was improved, and she seemed brighter. An examination of the knee-jerks at this time showed that the tendon reflex was absent. There was great atrophy of the muscles. The liver was found to be somewhat enlarged, and there was slight dulness under the left clavicle, but nothing definite was detected on auscultation. The lymph-glands were enlarged in both axillæ. The temperature at this time varied from 37.5° to 38° C. (99.5° to 100.5° F.), the thirty-first day from the child's entrance into the hospital, and continued at this height for the next week. During this time the child remained in about the same condition, but grew much weaker and showed more hebetude.

From the thirty-seventh day from the time when the chorea began the child grew much weaker, had incontinence of urine, refused to take her nourishment, and on some port wine being given to her vomited. She then was attacked with dyspnoea, which caused her to cry out loudly. On examination, dulness and diminished vocal resonance were found in various parts of the chest, and a few hours later she died.



These charts (Charts 29 and 30) show the child's temperature from the time when she entered the hospital until her death.

CHART 29.

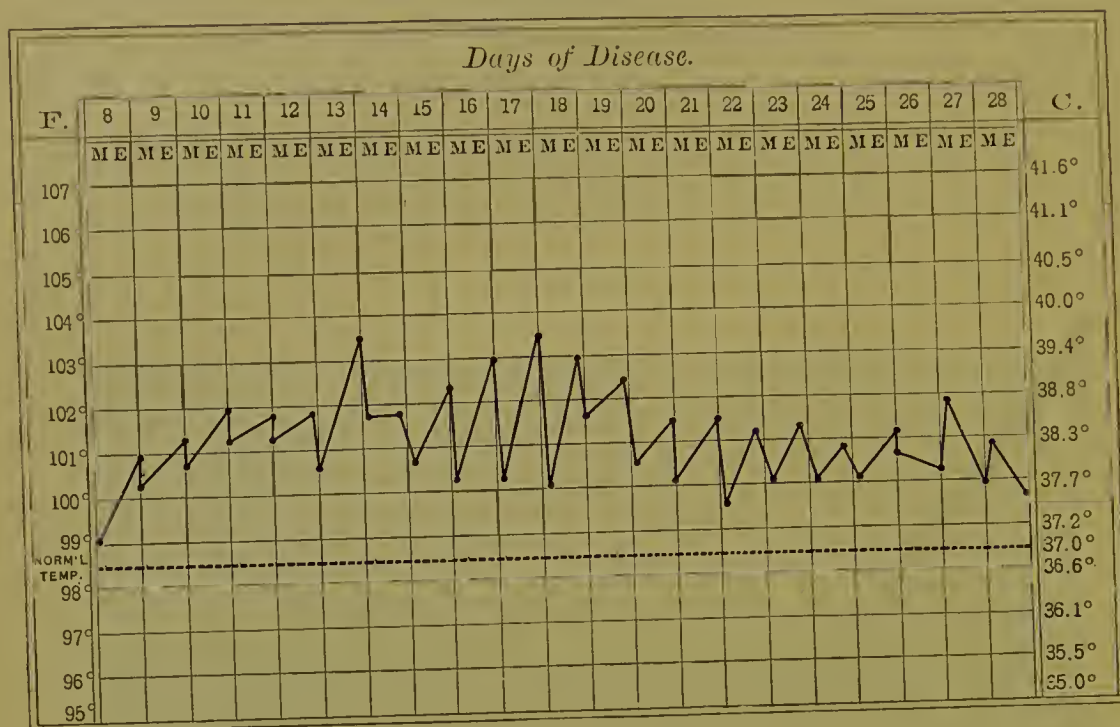
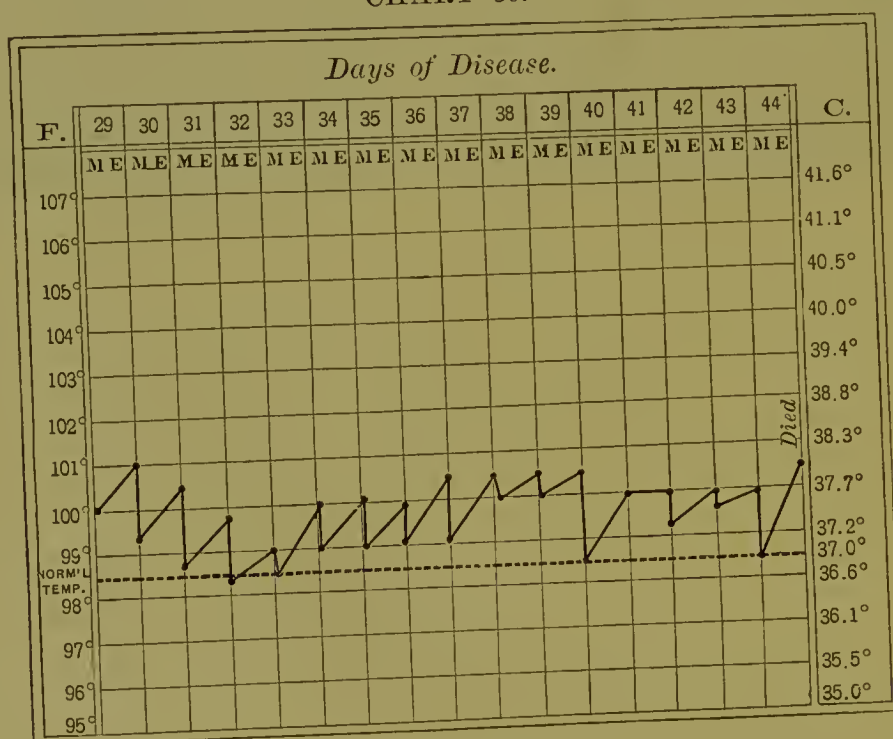


CHART 30.



The autopsy was made by Professor Councilman.

The head was not opened.

The peritoneum was normal.

The liver was enlarged, extending 4 c.m. (1½ inches) below the margin of the ribs.

In both pleural cavities there was a considerable accumulation of blood-stained fluid.

The anterior mediastinum was thickened.

The pericardium at the apex of the heart was adherent to the left pleura, and about this area the tissues were thickened and œdematous. The right lung was slightly adherent



to the pleura by comparatively late adhesions. The pleura of the lung was smooth, with the exception of the adhesions just spoken of. The lymphatics over the surface of the pleura were greatly dilated. The upper left lobe of the lung was congested and gave a sensation of small nodular masses in it. On section there was a distinct lobular consolidation throughout the upper lobe. The left lung was of a dark-red color, comparatively smooth on section, and somewhat solid. Muco-purulent matter could be squeezed out of the bronchi. The chief characteristic of the lung was the extreme dilatation of the interlobular spaces. The lymphatics all through the lung were visible. In the bronchi there was considerable œdematous fluid. The blood-vessels at the base of the lung were free. The bronchial glands were enlarged and reddened.

The left lung was not so adherent as the right. Over the posterior portion of the pleura there was a slight fresh pneumonia. The lung was somewhat compressed by the exudation of blood, otherwise it was in about the same condition as the right lung. The pericardial cavity was obliterated. The adhesions were easily broken down, except at the apex, where the pericardium was greatly thickened. Its surface was covered by a thick layer of fibrin and exudation.

The heart was enlarged. Over its surface was a dense layer of fibrin. At the apex of the left ventricle, at a point corresponding to the adhesion of the pericardium, the myocardium felt soft and had a whitish infiltration. Beside this, corresponding to the intraventricular septum, there was a line of rather firm, thick, whitish nodules. The interior of the right side of the heart contained moderately firm fresh clots. The surface of the myocardium on the right side of the heart was pale and soft. Along the free border of the auriculo-ventricular valve there were a few fresh vegetations. The left side of the heart was dilated, and the ventricle was thickened. The edge of the mitral valve was thickened and eroded, and there was distinct loss of substance in the thickened portion of the valve, which had irregular and eroded edges. The muscular substance of the heart was pale, with small whitish spots beneath the endocardium. These spots were very slightly elevated, and were more or less circumscribed. Similar spots could be seen in the cardiac muscles. The surface of the left auricle was thickened, and beneath the thickened area were numerous small whitish points. The aortic valves were intact, save for a few fibrinous deposits at the edge of the contact. The beaded row of elevations described on the surface of the pericardium corresponded to the course of the descending branch of the left coronary artery, and was probably due to thrombi with suppuration around them along the course of the artery.

The spleen was enlarged to the size of  $10 \times 8 \times 3\frac{1}{2}$  cm. ( $4 \times 3 \times 1\frac{1}{4}$  inches), and was comparatively soft. Over its surface were a few small adherent thrombus-masses. On section the Malpighian bodies were extremely prominent.

The mesenteric lymph-glands were enlarged and slightly softened.

The liver was large, the bile-ducts were free, and the gall-bladder was slightly distended. The portal vein was free. The surface of the liver was dark red. The lobules were prominent, and on section were slightly congested.

The pancreas was apparently normal. The suprarenal glands were normal. Both kidneys were of the same general size and appearance; in both the cortex was extremely pale, and the capsule was easily torn off. In the cortex there was a slight diffuse staining. The glomeruli were pale, but otherwise showed no change. In the lower ileum there was a slight enlargement of the follicles and of Peyer's patches. The glands at the root of each lung were enlarged and swollen. The left jugular vein was filled by a rather firm, slightly adhesive thrombus-mass, which extended downward into the subclavian vein and across this to the superior cava, into which it projected, and on the end there were a few soft fresh clots.

The anatomical diagnosis of this case was,—

1. Chronic pericarditis and mediastinitis.
2. Acute ulcerative endocarditis.
3. Thrombosis of innominate and left jugular veins.
4. Broncho-pneumonia.
5. Passive congestion and œdema of lungs.
6. Adhesions of pericardium.

7. Dilatation of interlobular lymphatics.
8. Acute pleurisy, right side.
9. Hydrothorax, both sides.
10. Acute spleen-tumor.
11. Occlusion of descending branch of left coronary artery.
12. Acute swelling of bronchial and mesenteric glands.

Cultures made from various organs showed the presence of streptococci, but not of pneumococci.

**EPILEPSY.**—Epilepsy is presumably an organic disease of the nervous system in which the pathological lesion has not yet been determined.

The characteristic symptoms are attacks of unconsciousness with or without convulsions, with a great liability to a recurrence of these attacks through a long period of time. The transient loss of consciousness without convulsions which occurs in epilepsy is called *petit mal*, while the loss of consciousness with general convulsive manifestations is called *grand mal*. Convulsions precisely similar to those occurring in true epilepsy may occur in organic cerebral disease as the result of external traumatism or from other causes; such convulsions have been termed *epileptiform*. The term *Jacksonian epilepsy* is applied to localized convulsions which are the result of organic cerebral affections. These latter forms must not be confounded with true epilepsy.

It is important that a sharp distinction should be made between the convulsions of true epilepsy and the many reflex convulsive attacks which come from a variety of causes and arise from the hypersensitive condition of the infant's nervous system. These reflex convulsions so closely resemble the convulsions which occur in epilepsy that the great importance of distinguishing between the two diseases can hardly be exaggerated. In the infant's rapidly growing brain the irritability of certain motor centres is physiologically far greater than in later childhood and in adult life. This irritability is the source of nervous explosions produced by many causes often slight in their nature, and it is impossible to differentiate these explosions by their clinical symptoms alone from the convulsive attacks of epilepsy.

**ETIOLOGY.**—It is usually granted that the initial lesion of true epilepsy lies somewhere in the cortical motor centres of the brain, and that the epileptiform convulsion is an irritation of these centres. True epilepsy may of course originate in early infancy, and does so in a large number of cases. Whether, however, infantile convulsions may be the cause of epilepsy is a very different question. The fact is that we do not as yet know what produces epilepsy. The various etiological factors which are usually cited, such as fright, injury, and dentition, probably have nothing more to do with the production of the disease than to precipitate its development in an individual who is already predisposed to it. Inheritance as a cause of epilepsy will presumably, in the future, hold a much less prominent place than has been granted to it in the past.

There is no good reason for believing that reflex convulsions in themselves ever lead to true epilepsy. It is of considerable importance that we



should be able to allay the natural alarm of parents by telling them, after the convulsions have ceased for a sufficient time to allow us to say that they are not epileptic, that there is no chance of their having produced an epilepsy which will develop later.

SYMPTOMS.—Epilepsy may begin in infancy or at any time throughout childhood, but a frequent time for its development is at puberty.

The *petit mal* may exist in different degrees of severity. In the mildest form, which may often pass unnoticed unless the attendants are especially on the watch for it, the child stops for a moment in its occupation, whether speaking, eating, or playing, while its eyes become fixed and it assumes a vacant expression. This condition may last for only a few seconds, when the child assumes its former occupation as though it had never been interrupted, and usually is not aware that anything has happened. In other cases this condition lasts a little longer, and slight twitching of the lower part of the face and of the extremities may occur. In other cases, again, the attacks are more severe, the child complains of being dizzy, staggers, has slight convulsive movements and turns pale, this condition lasting for a minute or more, and being quite marked, but without any total loss of consciousness. Momentary attacks of staggering sometimes occur alone in place of the attacks above described. At times these attacks of *petit mal* are the only manifestations of the disease, but in severe cases they are apt to be accompanied by occasional attacks of *grand mal*. They may occur as often as twenty or thirty times a day, or, on the other hand, they may be noticed only once in four or five days, and sometimes they are absent for longer intervals.

In the *grand mal* the attacks are of much greater severity. They are sometimes preceded for several hours by a feeling of malaise or general discomfort, but this is not always present. Patients sometimes have notice of the sudden onset of the attack, and such notice immediately preceding the convulsions and forming part of the attack itself is called the *aura*. This aura may be of different kinds. It is most commonly a sense of fulness or oppression in the epigastrium, from which something seems to rise into the throat, and unconsciousness supervenes. It may be, however, a pain or a sensation of numbness, tingling, or other form of paræsthesia in various parts of the body. Sometimes tinnitus is the first symptom. Frequently the patient has no warning whatever of the attack, but falls unconscious with or without a cry. The face becomes congested, and the eyes usually turn upward so that only the whites can be seen. After this follows the stage of tonic convulsions, which is sometimes so short that it is overlooked. Then come the clonic convulsions, which in typical cases are general, although the limbs on one side of the body are sometimes more affected than those on the other side. The movements of the limbs are apt to be very violent, the hands are clinched, the thumbs being flexed on the palms and the fingers closed over them. In many cases the patients froth at the mouth. In the more severe cases the children bite their tongues and pass their urine involuntarily. The duration of such attacks is usually five or ten minutes,

but one attack may succeed another with little or no intermission. When the attacks follow one another in this way for several hours the patient is said to be in the *epileptic status*, and his condition as regards life is very serious. After the convulsion ceases the child's breathing becomes stertorous and the limbs are relaxed. Later, and before consciousness fully returns, the child often falls into a deep sleep, and on waking has no recollection of the attack, but complains only of headache and of mental confusion. Attacks often occur in the night, and in this case may be overlooked, the only evidence of them being that the child has wet the bed. In certain cases where only nocturnal attacks have been present we often have reason to believe that the disease has existed for considerable periods before its presence was suspected. In some cases in connection with the attacks there is a desire to walk or to run, so that the patient must be closely watched. In this condition children may walk straight against an obstacle, though they are more apt to stop when something comes across their path. Sometimes they walk or run in circles.

The cases of paroxysmal running described by Bullard are at times the early manifestations of an epilepsy which will develop later, though they may also be only the symptoms of hysteria, chorea, and organic cerebral disease.

Epileptic children are liable to bursts of ungovernable anger and violence lasting for hours, in which they may tear and destroy things, bite the mother or nurse, and are apparently for a time under the influence of illusions and hallucinations.

The condition of patients between the attacks is in the lighter cases and in the beginning of the disease usually quite normal. As the disease progresses, however, there is a tendency to mental impairment, and in the more severe cases, in contrast to the lighter ones, we are apt to find some enfeeblement of intellect, which at times may go on to an advanced dementia.

It has been considered by some of the most acute observers that those cases in which petit mal exists in connection with the more severe attacks are more liable to mental impairment than those in which the grand mal exists alone.

DIAGNOSIS.—As the convulsive attacks occurring in epilepsy cannot be distinguished clinically from similar attacks due to other causes, we are forced to differentiate epilepsy from other diseases by carefully eliminating other causes for the convulsions. We must also wait to see whether the attacks will continue indefinitely, in which case they are more likely to be epilepsy. A very fair illustration of the difficulty which may arise in diagnosing infantile epilepsy is represented by these two infants whom I have had brought here to show you.

This infant (Case 341) was attacked at the age of ten months with general clonic convulsions. Previous to that time it had been mentally bright. It was then cutting the four upper incisors. One month later it again had a convulsion, the incisors having come through the gums. It is now two and one-half years old, and the convulsions have



continued, varying in intervals and in severity. The child is now somewhat impaired mentally, but there have been no other symptoms of cerebral disease.

The diagnosis of epilepsy can be made in this case, but this was not warranted at the time of its first convulsion, nor indeed for some time afterwards.

This second case (Case 342), eight months old, is, opportunely for your instruction, having a general convulsive attack. You see that it is unconscious; that the muscles of the face and of all the extremities are in active motion; and that the eyes are turned up. This is the third attack that it has had to-day. The lower middle incisors are almost through the gum; the gum is not swollen or tense, and shows no indication for lancing. We must therefore look further for the cause of this nervous explosion. There is no evidence of anything in the ear, and the normal temperature aids us in eliminating the prodromal convulsions of one of the acute diseases with high temperature. On coming out of the convulsion previous to this one there was no evidence from paralysis or stupor that any central nervous lesion had occurred.

So far as the clinical picture is concerned, this may be the beginning of an epilepsy, but the chances in a case like this are always that it is not one of epilepsy. The mother now remembers that she gave the infant last evening two or three beans which he managed to swallow. The case is probably one of reflex convulsions from gastric irritation.

(An emetic was given, the stomach was relieved of the beans, and the infant had no more convulsions.)

The diagnosis of epilepsy is made from a continuance of the attacks after a considerable period without evidence of any organic disease or marked irritation. When the child bites its tongue during the attack and goes to sleep after the convulsion, or when there is temporary mental impairment after the convulsion, we have good reason to state that the convulsions are due to true epilepsy, especially if no symptoms of organic brain disease coexist.

Epileptic convulsions are easily distinguished from hysterical ones by the presence of consciousness in the latter, at any rate to a considerable extent. Hysterical convulsions in children are not very common, and almost never exist without the presence of other symptoms of hysteria.

PROGNOSIS.—The prognosis of epilepsy for life is, on the whole, favorable, and epileptics may live for many years.

As regards cure, the prognosis in cases beginning in early infancy is very serious. When the disease begins at the age of ten years or later a certain number seem to recover, at least temporarily. Many authorities consider that true epilepsy is never cured, yet undoubted cases exist where no convulsions take place for years.

TREATMENT.—The child should be treated at once, in order to avoid continuous shocks to its nerve-centres. Much benefit results from early attention to general hygienic conditions, to diet, and to protection from nervous disturbances.

The management of these cases demands constant watchfulness and tact, so as to regulate the surroundings of the child in such a way as to avoid all source of irritation and nervous excitement. The diet must be regulated according to the especial indications for each patient. Slight gastric irritation apparently produces more serious consequences than irritation of any other part of the body. A vegetable diet is usually indicated, but where

the child does not thrive well on this it is advisable to give a certain amount of meat. Eggs are usually well borne.

The bromides in some form are, in my experience, the most useful drugs. It is often advisable in giving the bromides to change from one bromide salt to another, a greater effect being thus produced than by the constant use of one of them. Efficacious medical treatment depends more on the graduation of the doses, on the selection of the time for changing them, and on the determination of the intervals for administering them, than upon anything else. The best results in using the bromides are obtained by diluting the dose with a large quantity of water, 120 c.c. (4 ounces). As a rule, bromide of potassium has been found to be the most efficient and active of the bromides in cases of epilepsy. In giving the bromides it is well to begin with small doses, 0.12 to 0.24 gramme (2 to 4 grains), three or four times in the twenty-four hours, for the first year, and to double this amount for the second year. The dose should be increased gradually until the physiological action of the drug is noticed.

This treatment, at intervals of one or two weeks, should be carried on for long periods, and from six months to a year after the convulsive attacks have seemingly ceased.

This little girl (Case 343), who has been brought to the clinic this morning, is four and one-half years old. She was apparently a healthy infant. When she was ten months old she began to have convulsions, which were of a clonic type and infrequent at first, but when she was fourteen months old they became more severe and frequent. Since that time the convulsions have continued, and at one time she had fifty-four convulsions in forty-eight hours.

She was treated with bromide of potassium, beginning with doses of 0.12 gramme (2 grains) and gradually increased to 0.3 gramme (5 grains) four or five times in the twenty-four hours. Under this treatment the convulsions have become less frequent and her general health has much improved within the last year.

No other symptoms of disease have at any time been detected about this child, and the affection is simply represented by convulsive attacks followed by unconsciousness. Although the child shows considerable improvement at present, yet the probability is that she will never be entirely free from the epilepsy, and that as she grows older, especially as puberty is approached, the convulsive attacks may occur more frequently, and under these circumstances her mind may become more or less affected.

This child (Case 344), whom I have had brought to the clinic to-day to show to you as illustrating an extreme case of epilepsy, is three years old. She was healthy at birth, and remained so until she was two months old. At that time she began to have slight convulsive attacks, the cause of which could not be accounted for on a careful examination. During the earlier attacks she looked as if she were frightened. She would then scream, and become rigid and unconscious for about fifteen minutes, after which she would sleep three or four hours. These attacks occurred at all hours of the day and of the night. They have continued at irregular intervals, but are not now so frequent as in the first year and a half. During the first year she seemed as bright as any infant of her age, and developed normally.

She has been treated with the bromides, and they seem to have been of some benefit, but have not produced a permanent cure.

During the last year her mental condition has been much affected, and she evidently has a permanent injury of the brain produced by her epilepsy. She has never been able to sit alone or to bear her weight on her feet. She cannot feed herself, and she understands



very little that is said to her. The head is of about the normal size. The face and eyes have a vacant expression, and she has to be taken care of as though she were an infant in the early months of life.

In this case there is no history of epilepsy or of any especial nervous disorder in the family, nor of traumatism or of any serious disease which could have produced this nervous disturbance. We can only say, therefore, that it is a case of chronic epilepsy starting from some unknown cause and resulting in permanent idiocy.

This boy (Case 345) is ten and one-half years old. There is no history of epilepsy or of mental disease in his family. He has never had any especial diseases, but for the last six and a half years he has been attacked with convulsions occurring about once every three weeks. For the past six months the attacks are said to have been more frequent and severe. He at times has had as many as five in one week, and some of the attacks have lasted fifteen minutes or more. He states that just before one of these convulsive attacks he feels frightened and sick. He then loses consciousness and falls to the ground. The expression of his face is rather dull, as though the continued shocks to his nervous system were producing a certain amount of mental disturbance. He answers questions, however, readily and intelligently. A physical examination shows nothing abnormal.

## CASE 345.



Epilepsy, 6½ years' duration. Petit mal. Male, 10½ years old.

Since entering the hospital, two weeks ago, he has had a number of convulsions and has been closely watched, and there seems to be no doubt that the attacks are real and not hysterical. The attacks have usually occurred at night, but sometimes also when he has been up and about the ward. The attack is usually ushered in by a loud cry, and he is then found to be in a state of clonic convulsion. He froths at the mouth, but has not bitten his tongue. The attacks have lasted for about fifteen minutes, and have then been followed by coma and prolonged sleep for some hours. In addition to the other convulsive symptoms there has been much twitching of the face during the attack. The eyelids are usually half open, and the eyes rolled upward and inward. The pupils react only slightly during the attack, and the eyeballs are not sensitive to touch. As the convulsive seizure passes away, the reaction of the pupils gradually returns. During the attack there is no apparent sensation produced by pricking with a pin. Some of the attacks are preceded by restlessness and an attempt to get out of bed, so that he has to be restrained, and sometimes

a general feeling of uneasiness appears to precede the attack. He has been in this condition this morning, and has therefore been undressed and put to bed with the clothes simply thrown over him. As you now see him, he is evidently about to have an attack.

He is very restless, and has thrown back the bedclothes. You see that he now attempts to rise from the bed, and that his eyes are somewhat vacant and staring. These premonitory symptoms may simply represent an attack of petit mal, as a number of times they have gone no further, or may be the forerunners of a general convulsive attack such as is represented by grand mal.

(This especial attack happened to be represented by the form petit mal, and soon passed off without a convulsion.)

His pulse has been regular, and has varied from 70 to 90. His respirations have been about 24 in the minute. His temperature has been normal.

The treatment of this boy has been with bromide of potassium, but has not been followed by marked benefit. He is probably a case of chronic epilepsy which can never be cured, and which, according to Dr. Bullard's opinion after carefully examining him, will have to be taken care of in an institution for feeble-minded children.

This strong, healthy-looking boy (Case 346), seven years old, I have had brought to the clinic to-day to show you as another form of epilepsy. There is no history of organic nervous disease in the family. He was born after a difficult delivery, and on the following day had a number of convulsions, which continued at intervals for several days. They were of a clonic general variety, and were apparently relieved by small doses of bromide of potassium. During the first year, although the convulsions did not return, he had from time to time slight attacks, in which he turned pale and became almost unconscious. These attacks, however, lasted for only a few minutes. It is reported that during the first six months, although his physical development was fairly normal, he did not notice anything and seemed almost blind. After that time, however, his mental condition improved, although he seemed a little backward in comparison with other infants of the same age. During the first year and a half of his life his left leg seemed smaller than the right and was a little shorter, but no especial paralysis was noticed, and by the time that he was three years old no difference in the size of the limbs was detected. When he was one year old he was able to sit alone. His teeth were cut at the usual time. When he was two years old the measurements of the head showed that it was of about the normal size and the anterior fontanelle was closed. Towards the end of the second year he began to talk. When he was two and a half years old he had cut all his teeth and was well and strong, had a good appetite, and could walk well. He had, however, shown signs of mental disturbance. He was fretful, was subject to explosions of temper, and had to be carefully looked after to see that he did not hurt himself or the other children in the family.

When he was four years old he began to have convulsions of a clonic general type, occurring at night and ushered in by a scream. During these attacks he frothed at the mouth, was unconscious, had stertorous breathing, and after five or ten minutes would fall into a deep sleep from which he could not be aroused for a number of hours. On the following day he would be somewhat dull and fretful, but these symptoms would then pass away, and the convulsions would not return sometimes for a number of months.

As you see him to-day he is physically well developed. His mental impairment is, however, very evident; his eyes are not bright, he has a rather vacant, idiotic expression, and, although he has learned to read, he does not show as much intelligence as his brother who is four years old. Physical examination shows nothing abnormal.

This case illustrates a cerebral injury taking place at birth. This injury has left its mark on the brain in such a way that entire recovery will probably never take place. The convulsions are evidently epileptiform,—that is, they are caused by an irritation of some of the motor centres produced by the original cerebral lesion.

In a case of this kind treatment by drugs is usually without benefit. The attacks seem to be somewhat controlled by the bromides. There has been at times much constipation; when the constipation is excessive the attacks are more likely to occur, and it has been found that if the bowels are carefully attended to and the constipation thus combated he is in a better condition. It is a case in which much benefit can be obtained by mental



training, and he should be placed in some institution devoted to the training of feeble-minded children or in the hands of some expert in this branch of psychology.

**INSANITY.**—Insanity in children is very rare. In the ordinary forms of insanity no definite pathological lesion has been found which would account for the symptoms presented. Such changes as have been detected come very late in the disease and seem to be secondary. In parietic dementia, however, we find a special form of cortical interstitial encephalitis.

Instances of mania and melancholia at times occur. Hallucinations, which are a common symptom in the insanity of adults, occur in children usually in connection with the delirium of fever, or more rarely with epilepsy, as I have already described. Insanity is met with in children at any age; it is extremely rare before puberty, but then becomes more frequent.

The prognosis of insanity in children varies according to its form. Acute mania and melancholia are said to recover generally. True parietic dementia is never known to recover.

## LECTURE XXXVI.

## III. FUNCTIONAL NERVOUS DISEASES.

(Organic nature not yet shown.)

## (1) PROBABLY CENTRAL.

HYSTERIA.—HYPNOTISM.—CATALEPSY.—SIMULATED DISEASES.—INSOLATION.—CONCUSSION.—TEMPORARY AMNESIA.—TEMPORARY APHASIA.—ARRESTED PSYCHICAL DEVELOPMENT.—RETARDED SPEECH.—HEADACHES.—VERTIGO.—SENSITIVE SPINE.—TETANY.—PAVOR NOCTURNUS (Central).

TO-DAY, gentlemen, I shall speak of a class of cases which you are liable to meet with interspersed among the patients with definite diseases whom you are called to see.

These cases are called functional, and are represented by either a temporary suspension of, or a perverted use of, the normal physiological functions of the nervous system. We have at present no sufficient evidence to justify us in classifying these diseases as organic. These functional nervous phenomena play a rôle of considerable importance in early life, as they occur much more frequently at this period than they do in adults. The various functions of the nervous system in early life are in the process of development, and are not so perfected as they are in the more mature subject; in fact, they are in a state of unstable equilibrium: hence shocks of various kinds easily cause temporary disturbances which, not being grossly organic, can pass away after a period of rest.

This class of functional disturbances may be divided into nervous phenomena apparently resulting (1) from some affection of the nervous centres, and (2) from some irritation of the peripheral nerves.

I shall first speak of those functional diseases which are supposed to be of central origin. Of these hysteria is perhaps the most difficult to differentiate correctly and to understand, and I shall therefore begin with that disease.

**HYSTERIA.**—Hysteria is a functional disturbance of the cerebral centres represented, according to Möbius, by a state in which ideas control the body and produce morbid changes in its functions. The name is a misnomer, but it has been adopted so generally that we must use it for the present.

We know very little about the etiology of hysteria. Well-marked instances of the disease occur in early life, usually in the middle and later periods of childhood.

An inherited nervous organization or highly exciting surroundings, combined with a lack of proper home discipline, appear to present as likely a



field for the disease to develop in as any conditions, such as fright, which apparently, at times, directly lead to it.

The mere presence of emotional or imaginative conditions in children does not constitute hysteria. For the existence of the disease it is necessary to have definite symptoms, either a markedly disorganized mental state, paralysis, anæsthesia, or some serious loss of function (amaurosis, deafness, dysphagia).

SYMPTOMS.—The symptoms in this most protean of diseases are innumerable. Convulsions and paralysis are quite common, while dysphagia, amaurosis, and anæsthesia are met with only in the very severe cases, and are not often seen in America. Anæsthesia is especially interesting as representing a pure type of the disease, and is usually on one side of the body. Children perhaps only two or three years of age affected by hysteria will sometimes allow themselves to be pricked on the anæsthetic side of the face without wincing.

Hysteria in children as usually seen in America is marked by the emotional conditions of the child, and by the presence, in many cases, of a fixed idea relating to its own physical condition. The child believes that it cannot perform certain actions or functions, and hence does not perform them. There probably has often been in the beginning some real difficulty or disturbance of the performance of these functions, such as pain, which has passed away or which is not sufficient to produce the present condition.

The most common symptoms, aside from the mental condition, are (1) convulsions, (2) paralysis, and (3) anæsthesia.

(1) The convulsions are distinguished from those of epilepsy by the absence of loss of consciousness. The patient never seriously injures himself in falling, and does not bite his tongue. He does not sleep after the attack.

(2) The paralysis is often of the spastic form, and may be either hemiplegic or paraplegic. In this form the limbs are rigid and the knee-jerks are exaggerated. It may, however, be of the flaccid variety, with the knee-jerks diminished or absent. It is distinguished from the organic forms of paralysis by the normal reaction of the muscles to electricity, by the absence of atrophy, by the absence of any affection of the sphincters, and at times by the presence of anæsthesia.

(3) When anæsthesia occurs it is usually irregular in distribution, occurring in patches, or else it has the same distribution as in cerebral organic disease. It is often variable, changing more or less from day to day.

Although almost any symptom may occur in hysteria, yet the lack of uniformity in the grouping of the symptoms, and the combination of symptoms which belong to entirely different diseases, are of great aid in making the differential diagnosis from these diseases.

We sometimes meet with an exaggerated hysteria in children. The attacks are represented by screaming, running, jumping, and a feeling of being pulled about; they may last for hours, or for days; their duration,

however, is usually long,—at times, with intervals, over a year. No signs of organic disease are found in these cases; they seldom injure themselves, and are finally cured by moral influence, change of scene, and good hygienic surroundings.

Hysteria occasionally causes children to present symptoms of serious disease of the spine and joints. This most often follows some slight injury, but may occur spontaneously.

PROGNOSIS.—The prognosis in cases of hysteria is, as a rule, favorable.

DIAGNOSIS.—Generally, the diagnosis is not so difficult as in adult life, because the child is not able to control its sensations of pain and fear so completely as is possible with adults. In surgical cases, however, where hysterical affections simulate most closely organic disease of the joints, the diagnosis is often attended by extreme difficulty. The application of strong currents of electricity will usually show that the anæsthesia is not real.

A complete differential diagnosis of hysteria would occupy more time than I can give to the subject, and I have therefore merely outlined the general principles by which you are to be guided in diagnosing these cases.

TREATMENT.—The treatment of hysteria is to break up at once the harmful home surroundings, if such exist, and by means of gentle but firm compulsion to make the child understand that its symptoms are unreal. The various local symptoms connected with the digestion and general health of the child should be carefully treated, as the hysterical symptoms are often largely dependent on conditions of this nature.

This little girl (Case 347), ten years old, is about to leave the hospital, as she has recovered entirely from the disease for which she was brought here for treatment.

The history of the case is that her parents are living and well, and that there are a number of other healthy children in the family. This child had always been well until eighteen days before she entered the hospital. At that time she complained of headache, and on going to school returned feeling sick and apparently unable to speak. She is said to have been unconscious at times, to have had spasms, and to have been very restless at night. She evidently had had great lack of care in her home life, and had been given only poor food. She showed the evidence of this lack of care in the condition of her skin and her digestion on entering the hospital. A physical examination showed nothing abnormal in connection with the thorax and abdominal organs. The pupils were slightly dilated, but were equal and reacted to light. The knee-jerks were decreased. There was no ankle-clonus. She was apparently unable to walk, and she lay in bed taking no notice of anything, but winked her eyes if anything was thrust towards them. Her hearing did not seem to be especially impaired. She lay in bed in a very limp condition, with the legs drawn up in various positions. Her head kept rolling from side to side, and occasionally was retracted. When asleep her head was retracted so as to make nearly a right angle with the body. It was difficult to feed her, as she would not swallow. Her temperature was 37.2° C. (99° F.), her pulse 66, and her respirations 16. When being examined she cried out a great deal.

She was given plenty of good food, and in three or four days her condition was much improved. She took her food well, but was apparently unable to feed herself. A few days later she showed more intelligence, and on being taken up and dressed it was found that she could sit alone and could walk a little with support. On beginning to walk she threw her legs about wildly, but after being scolded she walked much better. At one time when she was sitting quietly in a chair the visiting physician came into the ward, and she imme-



diately allowed herself to slip from the chair and roll onto the floor, but evidently was careful not to hurt herself. She at this time cried out a great deal, but stopped when no notice was taken of her. She was still unable to speak, and, although she could sit up in a chair, apparently noticed nothing.

Nineteen days after entering the hospital she appeared much brighter, and began to take slight notice of what was going on about her. When questioned, she moved her lips as if about to speak, but made no sound. She continued to improve slowly, and a few days later said "sister," understood what she was told to do, and attempted to do it. She also walked three steps without being assisted. Some days later it was found that she would repeat almost any word that was said to her, but in a whisper. After this she improved rapidly and began to articulate fairly well, but slowly and with an effort. She also spoke voluntarily two or three times. She could not walk without assistance, as she would put her foot too far forward. She had been very much constipated through the whole attack, but at this time the constipation grew less. A definite training of the arms and legs was then begun by means of passive movements and massage. Under this treatment she has, as you see, greatly improved, and to-day, the thirty-fifth day from the time when she entered the hospital, has recovered completely. She speaks and walks, although she is still a little awkward.

I have concluded that this is a case of hysteria, as she has shown no definite symptoms of any other disease, and on account of the emotional character of her symptoms since she has been in the hospital.

**HYPNOTISM.**—Hypnotism is an artificial mental condition which can be produced in children as well as in adults. It is supposed to be a temporary abeyance of the powers of the higher cerebral centres. In the ordinary cases the child is thrown into a condition in which the consciousness of his external surroundings is lost. This condition in outward appearance closely resembles sleep, but is produced artificially and can be artificially removed. Thus, the sensation of pain can be temporarily abolished, at least to a considerable extent. For this reason it has been supposed that it might be useful in the treatment of cases requiring minor surgical operations. It has also been advocated by some physicians as a form of treatment in various diseases; but our experience at the Children's Hospital has proved it to be inefficient.

**CATALEPSY.**—Catalepsy is only a symptom. It denotes a condition, apparently of cerebral origin, in which, together with total or partial loss of consciousness, the limbs assume a peculiar form of rigidity called *waxy*, and remain for a considerable time in any position in which they may be placed. It occurs at all ages, but is very rare in childhood. The youngest case that I know of is that of a little girl three years old, reported by A. Jacobi.

The prognosis and treatment are those of the primary disease. There is no special treatment for a single attack.

**SIMULATED DISEASES.**—On the boundary-line between children who evidently are suffering from the need of judicious discipline and those who may be said to have the definite disease hysteria, is a class of cases in which *simulation* appears to play an etiological part. These children are usually in the later period of childhood, and seem to have such perverted functions of their nervous centres as actually to represent pictures of diseases which are easily proved not to be present. Deafness, blindness, pains of all

varieties, palpitation, dyspnoea, vomiting, spasmodic attacks of various kinds, and many other symptoms arise, and, may persist for long periods.

The best treatment for these cases is at once to show the child that you know his symptoms are unreal and of no importance.

You will remember the boy (Case 348), ten years old, who was brought by his mother to my clinic a few months ago with a history of convulsions which had been going on for two years, once or twice in a month. He was well nourished and robust-looking. The information was elicited from his mother that he never hurt himself when he fell down in a convulsion, and that the attacks followed attempts to make him do something which he did not wish to do. You may remember that I then suggested in his hearing that he should be sent to prison, and that he immediately fell on the floor, had a violent convulsion, foamed at the mouth, and was apparently unconscious. I then picked him up and told him that if he ever behaved in that way again he would surely be shut up in prison away from his mother. He instantly recovered, and has since been a reasonable member of society. You must be prepared to meet with all these different phases of nervous manifestations in early life, and learn to recognize to which class of nervous diseases they belong.

**INSOLATION.**—Heat-insolation, or heat-stroke, is a condition apparently represented by a functional disturbance connected with the cerebral circulation and produced by heat. This affection in varying degrees is of somewhat frequent occurrence in children, and is supposed to be accompanied by a hyperæmia of greater or less intensity of the meningeal blood-vessels. It is met with most commonly in the middle period of childhood, because at that age the child is most likely to be exposed to the influences which produce it.

The clinical picture of this class of cases is, as a rule, quite characteristic. The child has perhaps been playing on a hot summer's day somewhat more vigorously than usual, possibly romping with an older child of more highly developed nervous resistance, getting intensely excited, and greatly overtaxing its muscular strength. It may be that it has been exposed to the direct rays of the mid-day sun; or it may have been playing in some covered but heated and stifling place. The nurse of the child, noticing the extremely flushed condition of its face and head and its excited, sparkling eyes, takes alarm and hurries it to its home. Intense headache soon comes on, and in a few hours delirium may supervene. The skin is hot, dry, and reddened; there may be vomiting in the beginning; the carotids and temporal arteries throb perceptibly. The heart's action is violent, and the temperature is raised to  $38.9^{\circ}$ – $39.4^{\circ}$ – $40^{\circ}$  C. ( $102^{\circ}$ – $103^{\circ}$ – $104^{\circ}$  F.); the pulse is much accelerated, perhaps 140 to 150, and is full, but usually rhythmical. The conjunctivæ are congested and the pupils contracted. Photophobia to a greater or less degree is almost invariably present. Beyond this there may be no symptoms except a slight amount of muscular twitching, and in some cases a convulsion may occur if the temperature runs as high as  $40^{\circ}$ – $40.6^{\circ}$  C.



(104°–105° F.). The temperature, however, in accordance with the rule in this disease as in others which occur in children, does not always produce the same or equally severe symptoms. Thus, a temperature of 39.5° C. (103° F.) may in one individual give rise to marked nervous symptoms, while in the next child that you are called upon to see of the same age and with the same disease, a temperature of 40° or 40.6° C. (104° or 105° F.) may produce no nervous symptoms whatever, beyond possibly a slight apathetic condition. Convulsions may occur as a very common form of nervous explosion where fever and disturbance of the cerebral circulation are present, but, as a rule, this symptom is absent.

PROGNOSIS.—Be careful as to the prognosis which you give in these cases. Although they often simulate closely a beginning meningitis, a disease in which the prognosis, as I have already told you, is unfavorable, yet they are very amenable to treatment, and should therefore be carefully differentiated from that disease. In very severe cases the children may, of course, die of insolation, as do adults.

DIAGNOSIS.—The diagnosis from meningitis is based upon the history, the milder grade of the symptoms, except the headache, and finally, in doubtful cases, the quick recovery and speedy disappearance of the fever.

TREATMENT.—The treatment of heat-insolation should be prompt and vigorous. A stimulating enema of salt, one teaspoonful to a quart of water, should first be given. The child should then be placed upon a bed protected by a rubber sheet in a cool, darkened room; a warm mustard pack should be applied to the lower extremities, and the neck and chest gently sponged with water at 25° C. (77° F.) for fifteen minutes out of every hour. Leiter's coil should be applied to the head with water at 5° C. (41° F.); bromide of potassium should be given, 0.3 gramme (5 grains) every hour for four doses; a little iced milk may be taken if the child cares for it, not more than one or two ounces at a time; and complete rest and quiet for at least twenty-four hours are usually indicated. The child should be watched carefully for some days and not allowed to play actively enough to get heated. Great care should be taken for the rest of the summer to protect the child from the direct rays of the sun, as after one attack the cerebral circulation remains in a very sensitive condition for a considerable period.

I have found in my notes an account of two cases of this kind occurring in my practice, which I shall report to you.

A boy (Case 349), ten years old, healthy and well developed, was perfectly well on the morning of August 20. The weather was hot and sultry. He played unusually hard with some older boys for several hours in the sun. He then went into the house at 3 P.M. with his face and neck intensely reddened. The skin was hot and dry, the blood-vessels were throbbing, and there was severe frontal headache. His temperature was 40.5° C. (105° F.); his pulse was 160, full and bounding, and the respirations natural. He complained of photophobia. He was put to bed in a darkened room and the bowels were moved freely by an enema of soap and water. His head and neck were sponged with ice-water. At 4 P.M. he was slightly delirious, but could be aroused; his temperature at this time was 40° C. (104° F.). Bromide of potassium in doses of 0.3 gramme (5 grains) was given at 4 P.M. and at

5 P.M. At 6 P.M. the headache was less, and he fell asleep. He awoke at 10 P.M., and the temperature was found to be 38.9° C. (102° F.). He then took some iced milk and a dose of bromide of potassium. Later in the night the temperature was found to have fallen to 37.7° C. (100° F.), and the pulse to be 100. On the following day the temperature was 36.7° C. (98° F.), and the pulse 90. He complained of slight headache, but there was no photophobia. He felt weak and drowsy, and was kept in a dark room all day, taking small doses of milk.

On the following day he felt well, had a good appetite, and was up and dressed.

On the next day after driving in an open carriage in the sun for an hour he had a headache of moderate severity, but no fever, and was free from headache and perfectly well on the following day.

During the next two or three years, although he did not have any recurrence of a severe character, from time to time during the hot weather he showed that his cerebral circulation was still in a sensitive condition, as slight exposure to the rays of the sun caused considerable headache.

The next instance of this kind was a boy (Case 350), five years and three months old. On August 24 the weather was hot and sultry. The boy was perfectly well during the day, and was not exposed to any especial excitement or exertion. He went to bed well. The night was hot, no air was stirring, and the room in which he slept was very hot, close, and oppressive.

The next morning he awoke at 5 A.M. with severe frontal headache; he was very drowsy, had no appetite, and his temperature was found to be 38.9° C. (102° F.). He was kept all day in bed in a cool room. At times he would cry out from the pain in his head. His mind was perfectly clear. A dose of bromide of potassium, 0.3 gramme (5 grains), was given at 6 P.M. His temperature at that time was found to be 39.5° C. (103° F.); the pulse was 140, full and bounding. In the night he became delirious and had to be closely watched, as he would jump out of bed and cry out with pain in his head. Cold compresses were applied to his head during the night, and on the following day the symptoms were much relieved. Half a Rochelle powder was given in the morning and repeated in two hours. This was followed by a free movement of the bowels. He felt dull and complained of headache, but at 6 P.M. the symptoms were much relieved, and his temperature was found to be 36.7° C. (98° F.) and his pulse to be 100. On the following day he was reported to have had a good night and to have awakened perfectly well.

For two or three years following the attack he was liable to have attacks of this kind, either from undue exposure to the sun or at night if the room in which he slept happened to be ill ventilated and hot.

**CONCUSSION.**—By concussion we mean clinically a group of symptoms following some physical shock, with its resulting traumatic irritation of the nervous centres. I have met with a number of instances of this nervous phenomenon.

One was the case of a boy (Case 351), four years old, who fell from a table to the floor. I saw him an hour later, and found that his skin was cool, and his pulse slow, 60, and that he was nauseated and had been vomiting. No evidence of traumatic injury or proof of an organic lesion could be found. After a few hours the symptoms gradually improved, and he was perfectly well on the following day.

These indefinite symptoms are usually ascribed to the brain as the seat of irritation.

The treatment of a case of this kind is simply by perfect rest and quiet in a darkened room, with hot applications to the feet and abdomen, and small and repeated doses of stimulants given by enemata until the stomach is able to retain them, the treatment being continued until the circulation is normal and the pulse strong.



The next instance of this kind was the case of a little girl (Case 352), sixteen months old, whom I saw in consultation with Dr. Townsend.

The child was perfectly well, and was not of an especially nervous temperament. She could speak a number of words and could walk. While sitting in her baby-carriage one day at the top of a hill, another child took hold of the carriage and pushed it with great rapidity down the hill. The carriage was tipped over, and the child was thrown out on the sidewalk, apparently striking on her head. She was unconscious when she was picked up, but no signs of injury beyond a slight bruise on the right side of the head could be detected. There was no vomiting. Her extremities were cold. Consciousness soon returned, and nothing abnormal could be detected on a physical examination. The pulse was 120, and the temperature 36.6° C. (98° F.). After the accident she had no appetite, and became very cross and easily frightened. The bowels were moved regularly, a slight amount of blood, however, appearing in the first discharge which occurred after the accident. It was difficult to make her go to sleep, and she would wake up screaming and at times not knowing any one. For several days she could not make use of the words which she ordinarily did, and did not recognize her father, but was afraid of him, while before the accident she enjoyed playing with him. She seemed to have the same fear of a number of other people in the house, but did not show any symptoms of fear when being examined by Dr. Townsend or by me, although we were entire strangers to her. These symptoms continued for some weeks, gradually subsiding, and were not accompanied by any other abnormal condition, such as a rise of temperature. The child recovered entirely.

The treatment of the case was simply to keep her in a rather dark room separate from everybody but her mother.

I have here to-day a number of instances of other central and functional diseases to show you.

As the causes of these nervous manifestations are manifold, and as we know nothing about their pathology, I can best describe their symptoms and treatment by describing individual instances of the various cases of this kind which have come under my observation.

**TEMPORARY AMNESIA.**—Here are two cases which belong to a class of nervous disturbance which is represented by temporary amnesia.

This child (Case 353), now ten years old, was playing when he was nine years old on an asphalt tennis-court with some older boys. One of the boys threw him down on the hard court so that he struck the back of his head. He got up, but felt dizzy, so that he did not attempt to play any more, but sat looking at his playmates and occasionally making foolish remarks. This finally attracted the notice of his companions, who took him home. He was put to bed, and seemed drowsy, but did not have any nausea or any other symptoms, except that he could not remember anything, even that he was present at the wedding of his aunt on the previous day. He articulated plainly and spoke naturally. After sleeping for about twelve hours he woke up with his memory perfectly restored, except that he had a very dim remembrance of what had happened to him. Since the accident his mental condition has been normal, and as you see him to-day he is a bright, well-developed boy.

The probable cause of his amnesia was a physical shock with resulting abeyance of function in the nerve-centres connected with memory.

This next boy (Case 354), thirteen years old, is a case of the same kind. While running, about six months ago, he struck his head against a tree. I saw him three hours later. He had walked home, but was a little nauseated, and was put to bed. I found that he had partial loss of memory and was drowsy, but that he had no especial pain. He was perfectly well on the following day, and is, as you see, an intelligent boy, without mental disturbance of any kind.

**TEMPORARY APHASIA.**—An instance of suspension of the cerebral function connected with the elaboration of words is illustrated by Demme's case (Case 355) of

a child six years old, who, previously well and bright, suddenly lost the power of speech. This phenomenon occurred during an operation for talipes, which was being performed without an anæsthetic. After the operation the child was perfectly well, but was unable to elaborate words until the ninth day, when she began to use the one word "mamma" for everything that she wanted to say. She then gradually increased her vocabulary until the twenty-first day, when her aphasia disappeared entirely, and she developed mentally and physically in a normal manner.

**ARRESTED PSYCHICAL DEVELOPMENT.**—Arrested psychical development is a term used in speaking of an apparent lack of mental growth which is sometimes met with in infancy. So far as we know, it is a functional and not an organic condition of the brain. Infants with this affection develop both mentally and physically for a variable period, perhaps five or six months, and then continue to develop physically but cease to develop mentally. This condition lasts for a variable period of months, when they begin to develop mentally again, and, although for some time they are backward in comparison with other children of their age, they finally show no trace of an abnormal mental condition.

Arrested psychical development seems to be rather commonly associated with rachitis, and may also occur in the course of severe illnesses, but nothing else is definitely known concerning it.

**RETARDED SPEECH.**—At varying periods during the latter part of the first year and the beginning of the second year infants begin to make their first attempts to speak. By the middle of the second year they are usually able to communicate their ideas by means of short, broken sentences. In the third year most children speak quite plainly, though they do not correctly use the prepositions and adverbs until some years later. When during the second year the power of speech does not develop with the usual rapidity, it is spoken of as retarded speech.

This lack of power to speak may be from a simple lack of development of certain portions of the brain, or from organic or functional cerebral disturbance. It may also arise from abnormal conditions outside of the brain. The cases which are caused by a lack of development may be of congenital origin, or may be due to an arrested cerebral development produced by a number of causes. These causes are usually connected with some serious interference with the cerebral growth, such as a severe illness. The organic aphasia is like that produced by some such organic lesion of the brain as exists in cases of cerebral paralysis. It may also be connected with the condition of idiocy. The functional aphasia I have already described. It may be produced by many causes, among others the infectious diseases. A child may for a time during a severe illness, and after convalescence has been established, apparently be unable to use the words that it was accustomed to before the illness. I have in a number of cases, however, noticed that the child speaks better than it did before the illness.

Retarded speech may also be caused by such physical defects as disease of the ear resulting in deafness, and from such a physical malformation of the mouth, palate, or vocal cords as to render articulation impossible.



In this connection stammering may be spoken of as a cause of retarded speech.

When a child of this kind is brought to you to decide why it is unable to speak, you should carefully investigate the previous history. In this way you can eliminate organic disease of the brain by means of the absence of the usual symptoms of such disease, especially hemiplegia, and by ascertaining that the child has not had any disease sufficiently severe in its character to interfere with the development of the centres of speech. After determining that the child is not an idiot, you should make a physical examination of the ear and mouth. If, on examining the ear, you find that the child is deaf, you will at once have a good reason for his not being able to speak. Even where young children have learned to speak fairly well, if they later become deaf from a disease like scarlet fever they are very apt to become mute also. Where such lesion of the ear has occurred before the child has learned to speak, he almost invariably is found to be a deaf-mute, although there may be no defects in articulation or in his mental condition. It is seldom that any defect in the mouth or throat is found which interferes with articulation, except in cases where very extensive lesions are present, such as cleft palate, and sometimes enlarged tonsils combined with a high-arched palate and a large adenoid growth. The tongue-tie which the parents usually consider to be the cause of the retarded speech is seldom present. Where no symptom of organic, functional, or developmental cerebral disease exists, where there is no physical deformity, and where the child hears well and seems bright and well developed in other ways, you can, as a rule, assure the parents that the speech is merely retarded and will probably develop later.

**HEADACHES.**—When pain in the head occurs in early life it is to be regarded more seriously than when it occurs at a later period, as it is more apt to indicate some grave central lesion. The various forms of organic headache which arise in children can be spoken of best as symptomatic of the various diseases in which they occur.

There also appears to be a type of headache which occurs in the later years of childhood irrespective of any disease and often unaccompanied by nausea. These headaches, as a rule, are not of serious import, and are usually classed under the term functional. They occur irregularly, and may be in any part of the head. They are often so severe that the child has to lie down. The intervals between the attacks are variable, and the length of the attacks varies from two or three hours to a day. Of these functional headaches the most frequent form in children is that due to anæmia.

Although in many cases headaches are caused by an improper regulation of the diet, yet there is evidently some other cause which we do not recognize in their production, as with exactly the same diet for many months a child will show no symptoms whatever of headache. In like manner, although we know that headaches in children may depend upon constipation, yet this class of cases occurs whether constipation is present or not.

Migraine also may exist in children, and is characterized by severe pain in the head, sometimes unilateral, sometimes bilateral, accompanied by nausea, dizziness, and generally vomiting. The attacks occur at irregular intervals, and usually last the greater part of a day. They may be brought on by apparently slight causes, such as over-fatigue or very mild indiscretions of diet, in those predisposed to them. These headaches are markedly hereditary.

Although all these forms of headache are ordinarily very intractable to cure, especially where no bad hygienic surroundings exist which might account for them, and where the child does not lead a sedentary life, yet, as a rule, the attacks have a tendency to lessen and disappear as the child grows older.

The treatment of these headaches is usually unsatisfactory, as the attacks seem to arise from some functional disturbance which, irrespective of any cause that we can ascertain, resists the best known hygienic and medicinal treatment. Strict directions should be given as to exercise and food. A change of air and scene is often a valuable adjunct to the treatment. In many cases the administration of fluid extract of ergot, as recommended by Dr. Russell Sturgis, has proved to be of benefit. I know of no better treatment for cases of this kind during the presence of an attack than absolute quiet in a darkened room and the use of bromide of potassium or bromide of sodium in sufficient doses to produce sleep, or at least to lessen the acute pain.

This boy (Case 356) is thirteen years old. He has usually been strong and well, active in his habits, and bright in his studies. When he was nine years old he had a light attack of scarlet fever. Up to that time he is said to have been healthy and never to have had any nervous disturbance during the dental period. Just before his attack of scarlet fever he had a severe headache. At first these headaches occurred only twice a year, but now he is attacked by them four times a year. All the headaches have about the same characteristics as the one from which he is now suffering. The pain is usually most intense in the top of his head, and extends to the front and back. The attacks generally last a week. In the second attack which he had, the pain did not last so many days as in the first and those which have occurred later, but he was left in a rather weak condition afterwards, so that he could not walk. There was no paralysis of the legs.

Yesterday he went to school as well as usual, but soon began to feel pain in his head, and had to return to his home. The headache has continued, and to-day, as you see, he cannot sit up, but has to lie down. He has no nausea, his appetite is good, and there are no special digestive disturbances, but he has a slightly coated tongue and a slightly raised temperature. The bowels are regular, and he complains of nothing but such severe pain in his head that he has to lie perfectly still. His diet has always been simple, and there are apparently no direct causes, such as the use of tobacco, to account for the attack. He has never shown any delirium, has always been perfectly conscious during the attacks, does not complain of any photophobia, and merely wishes to be let alone.

On examining him to-day you see that his temperature is 36.6° C. (98° F.), and his pulse 84, a little irregular, but not intermittent. On physical examination nothing of an organic nature is detected in the thorax or abdomen. The heart's action is somewhat irregular, and there is a slight murmur with the first sound at the apex, and an accentuated pulmonary second sound. He has never had rickets. He is rather anæmic, but of course is looking unusually pale to-day, as he is in the midst of one of these attacks.

In this case there may be some slight organic disease of the mitral valve, but, as the



child is well and strong between the attacks of headache, the murmur may be of functional origin. In either case the headaches can scarcely be accounted for by the cardiac disturbance, and can well be spoken of under the term functional.

In treating this case small doses of tincture of digitalis are indicated, on the supposition that some disturbance in the circulatory organs exists, evidence of which is given by the cardiac murmur.

We should, in examining a case of this kind, before speaking of the attack as functional, eliminate other possible causes. One of the most common causes in children, but which does not exist in this case, is pain caused by a strain of the eyes. In all cases of headache in children the cause of which is not evident, a careful examination of the eyes should be made, even though there be no symptoms which point to the eyes themselves.

As an illustration of a class of headaches the cause of which was formerly obscure, I show you this little girl.

She (Case 357) is twelve years old. She has suffered during the past two years with almost continuous headache, so that she has had to be taken away from school.

A careful physical examination of this child made by me two years ago failed to detect anything abnormal, except that she was suffering probably from the results of eye-strain. With the exception of the headache she has been well and strong, has had a good color, good appetite and digestion, and has simply been incapacitated from studying and reading on account of the pain in her head.

The child was examined by an oculist and was made to wear glasses. No benefit resulted, and until within a few weeks her parents supposed that she could not be cured.

Suspecting, however, that the eyes were really the source of the trouble, I referred the child to another oculist, who has made a change in the glasses, and the headaches have disappeared.

**VERTIGO.**—Vertigo at times occurs in children. It is a term applied to a condition in which the individual or the objects around him appear to be rolling about. It is called subjective vertigo when the patient himself seems to be turning, and objective vertigo when it is the surrounding objects that appear to move.

Vertigo has a variety of causes. It may be due to organic cerebral diseases, such as tumors of the brain, especially of the cerebellum, and to diseases of the ear and of the eye. It may also be due to circulatory disturbances, as in cardiac disease, and to gastric vertigo, as from improper food, also from tobacco and tea.

This boy (Case 358), thirteen years old, was referred to me by Professor Blake with the history that he had had a purulent otitis several years since, but that this had healed three years ago, leaving a condition of adhesions and cicatrices with considerable impairment of hearing, but with no trouble of the labyrinth nor any symptoms pointing thereto.

The child was always strong and well until he was seven years old, when he had the purulent otitis which Professor Blake treated. Three years ago he began to have attacks of dizziness accompanied by seeing white spots. He at times had nausea, but no feeling of spinning round or falling. He has since had this feeling continuously, and lately it has rather increased in severity. He has had no other abnormal symptoms, except that he feels somewhat weak. He sleeps well, his appetite is fair, and his bowels are regular. He has

good hygienic surroundings, does not smoke, and has never lived in a malarial district. He is a close student, and is not fond of active sports. He has never had any headache.

There is a strong probability that the vertigo in this case is caused by his drinking too much tea and by his sedentary life. I shall therefore simply have him stop drinking tea, and have told him to ride on horseback every day.

(Subsequent history.) Within a few weeks after the active exercise had been begun and the tea had been omitted from his diet, the boy ceased to have attacks of vertigo.

**SENSITIVE SPINE.**—Among the nervous symptoms of central origin is what is called sensitive spine, a case of which I have here to show you.

This boy (Case 359), thirteen years old, previous to one year ago was perfectly well, but since that time has complained of headache at times when studying, has lost his appetite, and has become constipated. He began to complain of his back at the same time that the other symptoms developed. The other symptoms have not been especially pronounced, but the pain in his back has grown progressively worse, and there is sensitiveness on pressure over the spine.

After I had first seen him and prescribed exercise in the open air and omission of school and of study, he improved for a time, and all the other symptoms disappeared, with the exception of the sensitiveness of the spine. Although at times this sensitiveness disappeared entirely, yet it has lately returned, and has been just as painful as in the beginning.

I therefore referred him yesterday to Dr. Lovett, who reports that there are no indications for mechanical treatment, that the spine is normal in every respect, and that Pott's disease can be positively eliminated. The tenderness of which he complains is one which we are accustomed to see in neurasthenic women. The probability is that, owing to his poor physical condition and his slight muscular development, his spine has had to depend on the ligaments to maintain it erect, that the sensitive condition and the pain are due to the strain which is brought to bear on them, and that this will disappear as his condition improves. We can therefore provisionally make the diagnosis of sensitive spine from debility, and I shall have him treated by massage, gymnastic exercises, and electricity. Some of these cases prove very intractable to treatment.

(Subsequent history.) Within a month after this treatment was carried out the boy recovered entirely.

**TETANY.**—Tetany is a term which is used to represent tonic intermittent muscular spasms without loss of consciousness. The condition is simply a symptom of nervous irritation, probably of central origin and not produced by organic lesions. This symptom is very apt to occur in cases of rickets, but it may occur in otherwise healthy children when they have various disturbances of the gastro-enteric tract. It is also met with in the course of many of the acute diseases, such as pneumonia.

The spasm usually affects the extremities and not the face, and is accompanied apparently by a certain amount of pain. The legs and arms are flexed and rigid, the hands and fingers tightly flexed, the thumbs usually beneath the fingers across the palms of the hands. In like manner the feet may assume various positions of flexion, such as that of talipes equinus or that of equino-varus. The length of time which the tetany lasts is very varied; it may be a few minutes or it may be hours or days.

The symptom in itself is not a serious one, the danger existing in the special disease which causes the tetany.

Tetany is to be distinguished from tetanus by the spasm of the masseter



muscles occurring early in tetanus, and by its being absent or occurring late where the child is attacked by tetany.

Cerebro-spinal meningitis is also to be differentiated from tetany by the heightened temperature, the severity of the general symptoms, and the convulsions and opisthotonos, which I have already described as characteristic of that disease, while these symptoms are not pronounced in connection with tetany.

The treatment is to be directed to the special cause of the disease in which the tetany occurs. Warm baths are indicated for the relief of the spasm, and bromide of potassium is the most efficacious drug in cases of this kind.

**PAVOR NOCTURNUS** (Central).—The night-terrors of children may occur from a variety of causes, and should not be considered as one disease, but as a symptom of a number of diseases. Any nervous disturbance, whether central or peripheral, may produce so profound an impression on the sensitive cortical cells of the brain that the child's sleep may be disturbed by a cortical irritation.

The special form of pavor nocturnus which may be considered central has occurred in this boy (Case 360), six years of age, who has been brought for advice to the clinic this morning. He has always been a delicate, thin, pale child, not caring much for open-air exercise, but inclined to remain in the house and to be read to or to have exciting stories told to him. His appetite is poor. He is mentally bright and precocious. Otherwise he appears to be well, and shows no signs of any organic disease. Last evening he was allowed to sit up rather later than usual, and a number of terrifying stories were told to him. He went to sleep as usual, but in about an hour waked up screaming. He was found sitting up in bed looking terrified. His eyes were staring at some invisible object, evidently a picture in his brain and not a reality; he was pointing at this imaginary source of his terror, and kept repeating that it was a black dog. It was impossible to pacify him for about ten minutes, and he did not recognize his mother during the attack. He then became more quiet; the wild look passed from his eyes; he recognized his mother, and soon lay down and went quietly to sleep. The cause of this attack, which is typical of the central form of pavor nocturnus, was evidently undue excitement of the cells of the cortex in a bright, nervous child before going to sleep. The treatment of a case of this kind is to accustom the child to more exercise in the open air, to prevent his reading anything but the most ordinary and simple books, and to have no stories whatever related to him.

## LECTURE XXXVII.

## IV.—FUNCTIONAL NERVOUS DISEASES.—(Concluded.)

## (2) REFLEX.

PAVOR NOCTURNUS (PERIPHERAL).—DENTAL REFLEX.—REFLEX NYSTAGMUS.—REFLEX OF THE EAR.—REFLEX OF THE LARYNX.—PAROXYSMAL GASPING.—REFLEX OF THE LUNG.—REFLEX COUGH.—REFLEX OF THE HEART.—REFLEX OF THE STOMACH.—REFLEX OF THE BLADDER.—REFLEX OF THE VAGINA.—REFLEX OF THE RECTUM.

PAVOR NOCTURNUS (Peripheral).—At the last lecture, gentlemen, I showed you a case of pavor nocturnus in which the symptoms were evidently of central origin. To-day I have to show you a little girl three years old who also has attacks of pavor nocturnus.

The child (Case 361), as you see, is robust-looking. She is said to be always well and strong; to have a good appetite; not to be nervous or excitable; to be fond of playing out of doors, and not to care to have stories told to her. Her mother also states that she is constipated, and that she has a tendency to overload her stomach. She has had disturbance of sleep for some time, and last night she had an unusually severe attack of pavor nocturnus. She had eaten a very heavy supper, and on going to bed she immediately fell asleep, but soon began to be restless, to throw herself about, to groan, and to grind her teeth. A little later she woke up screaming, and apparently had a certain amount of dyspnoea. She did not recognize her mother, but sat up in bed looking very much frightened and clutching at her throat. Her mother made her drink some warm water, which produced copious vomiting. She then became rational again, recognized her mother, and soon lay down and went to sleep. She has no recollection of these attacks on the following day.

This is evidently a case in which the irritation is of the terminal filaments of the pneumogastric nerve in the stomach, causing reflex symptoms of the nervous centres to such an extent that the child is terrified and feels as though she would stifle.

It is a case, therefore, of peripheral pavor nocturnus, and should be treated by moderating the diet and allowing the child to have only a light and digestible supper. You see that the two classes of cases are distinct and that their treatment is entirely different. You will also often meet with a mixture of both of these forms in which it is not possible to make a clear distinction between them.

I have collected a number of cases to show you which represent some other illustrative types of reflex nervous symptoms.

DENTAL REFLEXES.—The twitching which occurs in children at the time when a tooth is the apparent cause of a certain amount of discomfort and fever should be referred to here as a significant illustration of nervous phenomena from reflex causes. The cases are numerous, but scarcely of sufficient importance to report. In certain instances, however, convulsions of a reflex nature occur at this time and cease when the tooth has assumed its place above the margin of the gum. I have also met with some interesting cases of local oedema arising during the period of dental irritation.



One of these cases was a male infant (Case 362), fifteen months old, who some months previously, while cutting one of the second molars, had an attack of œdema of the hands, which was not accompanied by irritation or any other symptom, and which passed off after a few hours.

This same boy when the canine teeth were about to come through the gums was again attacked by œdema of the face. This local œdema, as in the previous instance, disappeared quickly.

At times I have seen a local œdema attacking one eyelid, so that the eye could not be closed.

Although we cannot say that the irritation from the teeth is necessarily the cause of these conditions, yet they so often arise during the dental period, and not during other periods of childhood, or before the fourth or fifth month, that we can at least say that in individuals of an excessively nervous temperament the irritation which evidently arises in certain cases when the teeth are developing may be sufficient to cause a nervous explosion, which in this sense may be spoken of as of dental origin.

These are only a few instances of the reflex disturbances which occur during the period of dentition, and I shall speak of the subject as a whole under the heading of difficult dentition (page 794).

**NYSTAGMUS.**—By nystagmus is meant a peculiar rhythmical oscillation of the eyeballs, usually from side to side.

It may be produced by many causes. It is sometimes dependent on organic disease of the brain, and sometimes it arises from local diseases of the eye. In certain cases it is reflex from various peripheral stimuli.

Nystagmus of reflex origin is not a very uncommon symptom in young children. I have notes of two cases, brothers, who during the dental period showed this oscillation of the eyeballs with no other symptoms. Complete recovery resulted when dentition was concluded.

This child (Case 363), three years old, has, as you see, nystagmus. She is rachitic, and did not walk until three months ago. She shows no signs of organic disease, and will therefore probably recover from the nystagmus when the rachitis is cured.

**REFLEX OF THE EAR.**—The reflex connection between the roots of the teeth and the membrana tympani by means of the otic ganglion produces the well-known reflex earache which occurs during the dental period. This phenomenon I shall speak of more fully later (page 795).

**REFLEX OF THE LARYNX.**—In certain cases reflex symptoms occur in the larynx. This condition is usually noted during infancy rather than in childhood. The affection has been called *laryngospasmus*, and, although it is more usual for it to occur in rachitic children than in others, it is not necessarily confined to rachitis. It is not in my experience a very common disease, but when met with it is very characteristic.

The infant is suddenly attacked by a spasmodic contraction of the larynx. This condition may be precipitated by various causes, such as fright and excitement. I have also seen it produced by various peripheral irritations, such as those of the nose. At times the attack is so severe

that the infant becomes unconscious and cyanotic. The attack lasts for only a few minutes, and on recovery the infant seems as well as ever. There does not seem to be an inflammatory condition connected with this disease, and apparently it is purely of a reflex nature. In some cases a crowing laryngeal sound will frequently precede and often succeed the more severe stage of the attack.

The prognosis in cases of laryngospasmus is, as a rule, favorable, although very weak infants may die in an attack.

As the infants are usually delicate and of a highly nervous organization, the treatment should be directed to improving their general health and to protecting them from nervous excitement until they have attained an age when their nervous system is less irritable and is in more stable equilibrium. During an attack the treatment is to endeavor to produce relaxation of the spasm by peripheral irritation elsewhere. This is usually done by showering the child on the chest and face with cold water and lightly slapping the back.

Among a number of cases of this kind which have come under my notice was this one :

A boy (Case 364), one year old, had always shown a nervous temperament and had had a number of convulsions when he was cutting his first teeth. With the exception of a light attack of epidemic influenza, he had been well and strong. Following the attack of epidemic influenza, in which the nasal symptoms were prominent, he was left with a very irritable naso-pharynx. He then began to have attacks characterized as follows :

Whenever the nurse, while giving him a bath, attempted to cleanse his nose, no matter how gently, he would immediately gasp, have a catching sound in his breathing, become rigid, draw himself back sometimes almost to the position of opisthotonos, become unconscious and cyanotic, and then after a few seconds the spasm would pass away and he would seem perfectly well again. These attacks continued for some months without apparently harming him, and they then grew less frequent and passed away entirely.

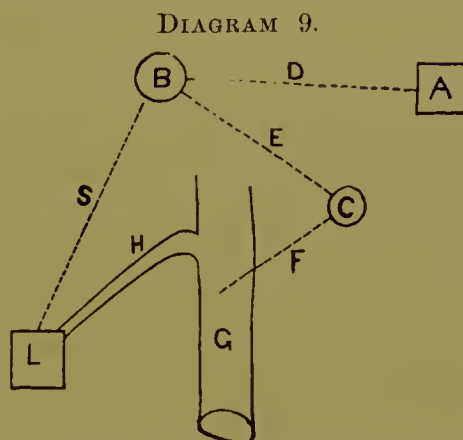
Two other cases, in which the attacks were similar to the one which I have just described, were of infants in their second year. In these cases the attacks were usually brought on by excitement and when the children were thwarted in anything which they wished to do. In addition to the symptoms which I have just described, there was in the younger infant a crowing laryngeal sound just as the attack was taking place and for a few inspirations after it had ceased.

As additional examples of reflex phenomena of the larynx having their origin in the ear may be mentioned the hoarseness which sometimes accompanies the impaction of cerumen in the ears, and which disappears almost immediately after the removal of the mass. Professor Blake reports a case where a persistent laryngeal cough of several months' duration was immediately relieved by the removal of a bead from the external auditory canal. These cases, as well as one of reflex cough (Case 368) which I shall presently report to you, can be explained by means of this diagram (Diagram 9, page 749), which shows the reflex connection between the ear and the larynx.

The irritation of the sensitive fibres of the auriculo-pneumogastricus distributed in the meatus and to the membrana tympani is reflected along the motor fibres of the superior laryngeal nerve, exciting in the larynx the



act of coughing by causing contraction of the crico-thyroid muscle. Where the original irritant, either in the meatus or in the membrana tympani, by its continued presence involves the vaso-motor fibres associated with the auricular nerve, they conduct their impression to the ganglion of the pneu-



Reflex connection between the ear and the larynx. A, auditory canal, membrana tympani, and middle ear; B, second ganglion of vagus; C, first cervical ganglion of sympathetic; D, auriculo-pneumogastric nerve; E, sympathetic fasciculus connecting B and C; F, nervi molles, vaso-motor connection with external carotid; G, external carotid; H, laryngeal artery; S, superior laryngeal nerve; L, larynx.

mogastric, and thence it is deflected through a sympathetic fasciculus proceeding from it to the first cervical ganglion. This again through the nervi molles carries the impression to the external carotid artery, and by its branches to the mucous membrane of the larynx, and as a result of reflected vaso-dilator impressions we may have congestion of the vessels supplying the mucous membrane of the larynx, and perhaps effusion from these vessels.

The detailed description and an illustrative diagram (Diagram 10) of the anatomical conditions underlying these reflex phenomena will be found in a later lecture (page 795).

**PAROXYSMAL GASPING.**—This boy (Case 365), eight years old, has always been delicate, and has evidently been ill fed and ill cared for. He is pale and thin, is of a nervous temperament, and has been overworked at school. Physical examination shows nothing abnormal in either his thorax or his abdomen.

The attacks from which he now suffers have occurred for the past month, and are growing shorter in their intervals and more severe in their character. The child, from being in a state of perfect quiet, suddenly becomes cyanotic, rolls his eyes up, stops breathing, gasps, and looks as though he were about to die. The attack lasts about half a minute, and he is then apparently as well as ever. Since he was brought to the clinic the intervals have been about fifteen minutes. The seizure is apparently a reflex irritation of the diaphragm, and could be classed under hysteria. These cases respond quite readily to good care, well-regulated food, and relief from the duties of school.

A boy (Case 366), twelve years old, with the following history was brought to me for advice:

He was of a nervous temperament. He was studious, and did not care to play with other boys, preferring to remain in the house and read. His appetite was pretty good; he was pale and rather apathetic. An examination of the thorax and abdomen revealed nothing abnormal.

Three weeks previous to my seeing him he began while sitting quietly in school to have paroxysmal attacks of gasping which he could not control. These attacks were of a much

milder grade than in the case which I have just shown you, but were quite as frequent. They did not occur when he was playing out of doors or exercising, and he had never suffered from palpitation or dyspnoea.

Treatment with various drugs, such as arsenic, nux vomica, and iron, given at different times and separately, had no effect upon the paroxysms. Taking the boy out of school, preventing him from studying or reading much, and making him go to the gymnasium and ride a bicycle, lessened the attacks in a few months, and he recovered entirely in about eight months.

**REFLEX OF THE LUNG.**—In young infants pulmonary attacks closely simulating the symptoms of asthma occur from gastric irritation of the terminal filaments of the pneumogastric nerve. They are evidently reflex in their character, and are promptly relieved by treatment of the stomach. They are spoken of under the term asthma dyspepticum. In cases of this kind it is usually found that the peripheral irritation either arises from the too high percentages of the solid constituents of the milk which is given to the infant, or is caused by the total amount of milk given being too great for the infant's gastric capacity.

The first symptoms noticed in these cases are the pallor of the infant's face, and a slight cyanosis around the mouth. The respirations then become quickened, and the infant is evidently in great distress. It becomes cyanotic, breathes very rapidly, and often looks as though it were about to die. On examining the chest the lung is found to be resonant, and there is nothing abnormal on auscultation except roughened respiration and a few sonorous râles.

An emetic will quickly relieve this condition, which disappears as soon as the stomach is emptied. The attacks are sudden and often recur. After the attack has passed off, the abnormal sounds heard in the lung are found to have disappeared completely, and the infant seems perfectly well again.

Another class of reflex pulmonary symptoms which has at times come under my notice consists of cases in which from some peripheral irritation elsewhere marked pulmonary symptoms simulating pneumonia arise.

This little girl (Case 367), six years old, is an interesting case of this kind. The first attack occurred at a time when she was having an exacerbation of an attack of subacute purulent otitis media. This happened one year ago, and she is brought to the clinic to-day apparently suffering from an attack of the same kind, so that I shall probably be able to illustrate its reflex nature.

As in the first attack, she has a heightened temperature, 40° C. (104° F.), and rapid respirations (60 in a minute). The *alæ nasi* are moving slightly, and she shows a certain amount of orthopnoea. Her face, as you see, is flushed, and she has a short, dry cough. She has a discharge of pus from both ears. On looking at this child you would naturally say that she had pneumonia. Evidence of this is given by the temperature, the respirations, the *alæ nasi*, the cough, and the orthopnoea. On examining the lungs, heart, and throat, they are found to be normal. The pulmonary symptoms are evidently reflex in their nature, as it is believed that in these cases the reflex symptoms are usually produced by the occlusion of the Eustachian tubes.

Now watch the effect of inflating the Eustachian tubes with the air-douche. You see that within a few minutes after I have inflated the Eustachian tubes her breathing has become normal in rate, 24 to 28 in a minute, the *alæ nasi* have ceased to move, the cough has disappeared, and the child can now lie down with comfort. The temperature will soon



begin to fall, and in place of the picture of a pneumonia which you saw a few minutes ago you see she is assuming the natural condition of a child with slight fever and a heightened temperature caused by inflammation of the middle ear.

**REFLEX COUGH.**—The nervous connection between the ear and the larynx gives rise at times when there is disease of the former, such as an otitis media, to a persistent cough which is evidently reflex, and which is relieved only by treatment of the ear. A very interesting case of this kind was for some time under my care in conjunction with Professor C. J. Blake.

A little girl (Case 368), four years old, had an attack of measles which was complicated by an otitis media. She recovered entirely from the measles, and seemed perfectly well, except that the perforation of the membrana tympani had not entirely healed. Somewhat later the cough began. Nothing was found to account for this symptom in the throat, lung, or larynx, except a slightly reddened appearance of the latter from coughing. The cough was intractable to all local treatment until the ear, which had been in the process of healing, again showed signs of increased inflammation. Whenever the ear was discharging, the cough ceased entirely. When Dr. Blake treated the ear and the discharge grew less, the cough began again, and there was an evident reflex connection between the larynx and the ear.

These reflex phenomena continued for some months, the child always coughing when the ear got better and ceasing to cough when the ear got worse. Finally, on the child's being taken to Switzerland and having an entire change of air, its general health was much improved and the reflex cough passed off. There has been no recurrence of this condition in the following ten years.

Where there is an irritation in the naso-pharynx a reflex cough often occurs, and is best treated by local applications to the pharynx and naso-pharynx. It is important for the physician to recognize this class of coughs, as he might otherwise be very unsuccessful in treating these cases. Many children are treated with drugs for a cough which is usually ascribed to bronchitis, where no physical signs of irritation can be found in the lung, larynx, or throat, and where the irritation is in the nose or the naso-pharynx. In place of the many drugs usually given in these cases, a simple spray in the nose is indicated.

**REFLEX OF THE HEART.**—I occasionally meet with cases of extreme palpitation in children where nothing organic can be detected, and where no cause, such as tea-drinking, is discoverable. The children are of a highly neurotic temperament, and are usually much influenced by exciting surroundings in their homes.

As an illustration of this class of cases I will show you this boy (Case 369), ten years old, who is subject to fits of great excitement brought on by the most trivial causes, such as preparing to go to school or to take a journey. For some hours before the proper time for starting comes he is apt to grow more and more agitated, thinking that it must be time to start. He will then often be seized with violent palpitation lasting for several hours and forcing him to lie perfectly still on his back. At these times his skin will be cool and pale, and his pulse weak and irregular. Nothing abnormal has ever been detected on an examination of the heart or any other organ.

(Subsequent history.) The attacks in this case lasted until he was twelve years old, and have never occurred since.

**REFLEX OF THE STOMACH.**—There are a number of reflex conditions connected with the stomach arising from different causes but represented by the same symptom, vomiting. Instances of this condition are those cases of vomiting which arise from irritation of the larynx and pharynx and which are cured by local applications made to these parts. There is another reflex gastric condition in which the vomiting is apparently caused by shock, probably affecting the abdominal sympathetic ganglia.

These cases can best be spoken of in detail when I describe the various affections of the stomach. I shall therefore merely refer to them here as instances of reflex functional disturbance.

**REFLEX OF THE BLADDER.**—Reflex spasm of the bladder occurs very frequently in young children. I shall consider it under the head of incontinence of urine when speaking of diseases of the bladder.

**REFLEX OF THE VAGINA.**—There is almost always a direct cause to be found for the reflex nervous symptoms which arise from vaginal irritation. One of the most common causes is the *ascaris vermicularis*, which at times gives rise to extreme and severe symptoms when it has migrated from the rectum. In addition to the local irritation, which causes the child great uneasiness, so that it cannot sit still and is continually moving its legs about, its temperament may be much affected. A child with this trouble is apt to be very fretful, to have violent outbursts of temper, to lose its appetite, and to grow thin. A case of this kind has lately come under my notice.

A little girl (Case 370), five years old, had the most extreme vaginal irritation. When I saw her she had been affected for several months and was in a very weak condition. At times the irritation seemed to be more than she could bear, so that she would lose all control of herself, would throw herself on the floor, and would have violent spasmodic contractions of the legs. Her sleep was much interfered with, and her whole appearance was that of a child suffering from some serious disease. An examination showed that the *ascaris vermicularis* was the cause of the vaginal irritation, and after a few days' treatment directed to expelling this parasite the child ceased to have any irritation and subsequently recovered entirely.

**REFLEX OF THE RECTUM.**—In certain cases reflex symptoms of a most exaggerated type are localized in the rectum. An instance of this phenomenon is this little girl, who, you will remember, was brought to the clinic several weeks ago.

She (Case 371) is four years old. She has always been remarkably strong and robust, and has never had any especial local trouble with the bladder or the rectum. She is, however, of an excessively nervous temperament, and is surrounded by exciting influences in her home.

A few months ago she began to have spasmodic contractions of the sphincter ani. When she attempted to have a movement of the bowels it frightened her, and she would clutch at any piece of furniture which happened to be near her and try not to have the movement. She would scream and cry out as if she were in much pain.

An examination under ether showed nothing abnormal in the rectum or sphincter, such as from pressure or from lesions, and the condition was apparently that of spasm simply.

For the last two weeks she has been treated by the daily dilatation of the sphincter ani



with bougies, the size gradually being increased. This has been followed by marked improvement, and her mother reports to-day that the trouble has passed away.

(Subsequent history.) The rectal spasm did not return in this case, but the child began to have incontinence of urine, from which she is still suffering two years later.

Another case which came under my care and which was a form of reflex connected with the anus was a little girl (Case 372), eight years old.

This child for a whole year was affected by intense irritation in the region of the anus, which prevented her from sitting down for any length of time and kept her in a continual state of irritability. Nothing could be detected during this period which caused these symptoms. No trace of intestinal parasites could be found, and nothing abnormal, either at the anal orifice or in connection with the bowels, was seen, the skin around the anus being in a perfectly normal condition.

The child was treated with bromide of potassium for several weeks, and recovered entirely.

What I have said concerning the reflex phenomena of infancy and early childhood must not be supposed to be a complete enumeration of these affections. Thus, various involuntary movements of the head in infants sometimes occur, such as *spasmus nutans* (antero-posterior movements) and *gyrospasm* (rotary movements).

## LECTURE XXXVIII.

## CONVULSIONS.—TREMOR.

**CONVULSIONS.**—Attacks of motor disturbances represented by continuous rigidity or contractions of one or more groups of muscles, lasting for a variable time and accompanied usually by unconsciousness, are designated convulsions. The term convulsion is applied to a symptom, and not to a disease.

Convulsions may be divided, as to their type, into (1) *clonic* and (2) *tonic*; as to their form, into (1) *general* and (2) *partial*; and as to the seat of irritation which causes them, into (1) *central* and (2) *peripheral*.

The *clonic* convulsion is an active spasmodic contraction of the muscles followed by immediate relaxation. The convulsions of epilepsy are illustrative of this type.

The *tonic* convulsion is a more or less continued spasmodic rigidity of the muscles. This type is seen in tetanus neonatorum.

The convulsive movements may affect the entire body and limbs, including the face, or they may affect only certain groups of muscles. Thus, they may be localized, as of one limb. They may be unilateral or bilateral.

The seat of irritation which produces the convulsion is very varied. Thus, it may be a lesion of the central nervous system or of the peripheral nerves; in the former case the convulsions are spoken of as *central*, in the latter they are termed *reflex*. Convulsions are much more apt to occur in infancy than in later childhood and in adult life, probably because the power of inhibition is not developed in the former. Not only, therefore, do we meet with convulsive attacks more frequently the younger the individual, but, as a rule, we are led to look upon these convulsive attacks as of much less import than in the older subject. The reason for this is that the causes of reflex convulsions in infancy are innumerable, and, as a rule, they do not result seriously, while in older children and in adults convulsions are almost always representative of some serious central lesion. Convulsions are in fact so common in infancy that they have been compared to the chill which occurs in the initial stage of many diseases arising in adults. It is a fact that the various acute diseases accompanied by high temperature, such as pneumonia and the exanthemata, are very commonly preceded by a convulsion, and that a chill is rare under these conditions in infancy. We must, however, not be misled by the frequency and comparatively benign character of convulsions in infancy and by the rule that they are not fatal. The convulsions of infancy may represent just as serious conditions as they do in later life, and we must always look upon them as a grave symptom until we can be sure, by eliminating serious organic lesions as a cause, that we are dealing



with one of the common and mild forms of this phenomenon. We must remember that the convulsion does not in itself show us whether it is the result of serious or of benign disease. The convulsions which occur from some organic lesion, such as cerebro-spinal meningitis, may differ in no way from those which arise from some simple cause, such as indigestible food. It is therefore well to speak of convulsions apart from the diseases in which they occur, and which I have already described.

We are frequently called to see an infant in convulsions where the convulsion is the first and only manifestation of the disease which is presented to us. The infant after a few signs of uneasiness suddenly becomes rigid for a second or two, makes a sound as though choking, the eyes turn upward and become fixed, there may be strabismus, the skin becomes somewhat cyanotic, and then the convulsive movements begin. The eyelids open and shut; the face and usually the head are drawn to one side; the fingers are clinched; the arms move up and down, as do also the legs. The back may at times be somewhat arched and the head somewhat retracted. The infant is apt to foam at the mouth to a greater or less extent; it is perfectly unconscious, and the breathing soon becomes stertorous. These symptoms after lasting for two or three minutes are followed by complete relaxation, an apparent state of coma, and sleep. The child on waking may be bright and well, or the convulsion may recur and continue for a much longer time, as in one of my cases, where an infant had fifty-two convulsions in twenty-four hours. There may be involuntary discharges of urine and of fæces.

I have had an infant brought to show you to-day who illustrates very clearly the fact that numerous convulsions do not necessarily lead to a fatal issue.

This infant (Case 373), six months old, is well developed, healthy, mentally bright, and has not had any convulsions since it was a month old. During the first two weeks of its life it had convulsions almost continuously.

This table (Table 105) gives the hours and intervals of the convulsions from 9 A.M. one day till 9 A.M. the next day. The attacks, as a rule, lasted only a few minutes.

TABLE 105.  
(Forty-one convulsions in twenty-four hours.)

<i>Time of Convulsions.</i>			
9 A.M.	12 Midnight.	2.42 A.M.	6.22 A.M.
11.35 "	12.35 A.M.	3.18 "	6.32 "
3.10 P.M.	12.40 "	3.25 "	7.10 "
3.50 "	12.50 "	3.42 "	7.40 "
4 "	1 Noon.	4.40 "	7.52 "
4.07 "	1.25 P.M.	5.08 "	8.07 "
4.28 "	2 "	5.20 "	8.17 "
4.40 "	2.12 "	5.30 "	8.25 "
5.55 "	2.25 "	5.45 "	8.35 "
6.13 "	2.38 "	5.55 "	9.02 "
11.50 "			

The most important, on account of their serious nature, are those convulsions which are of central origin, and I shall therefore first speak of this class. Convulsions of this nature may occur in any disease which is represented by a high temperature, such as insolation, meningitis, the exanthemata, pneumonia, or other diseases. In these cases the convulsions are produced either by the action of the high temperature on the motor centres of the brain, or by the direct action of the special toxic agent which is producing the disease. These convulsions, as a rule, are general, and are produced by the diffuse action of the poison. In this class of cases it is probable that there is an extremely hyperæmic condition of the blood-vessels of the central nervous system. The convulsions may also, in contradistinction to the supposed active hyperæmia of the blood-vessels and the high temperature, be produced by vascular stasis and a normal or subnormal temperature. This form of convulsions may occur in the course of pertussis or of cardiac disease. Again, convulsions are supposed to be caused by an anæmic condition of the blood-vessels of the brain, such as may arise from loss of blood or from exhausting diarrhœa. Such toxic agents as are represented by drugs of various kinds, as belladonna, may produce general clonic convulsions. Mental disturbance, such as sudden fright, has also been known to produce a convulsive attack. In all these classes of cases the convulsions may be partial and clonic instead of general, though the rule is, owing to the diffuse character of the irritation, that they are general. In addition to these convulsions which arise from a diffuse cause are those where, a local lesion having occurred in the brain, from morbid growths, embolism, thrombosis, hemorrhage, or any other cause, a disorganization of a portion of the brain has been produced. As these lesions are, as a rule, focal in their distribution, we are apt to have localized convulsions, as I have already explained to you in speaking of convulsions in cerebral paralysis, with their resulting hemiplegia.

A number of other diseases can also, by their direct effects, irrespective of the temperature which accompanies them, produce convulsions. Thus, convulsions occur not uncommonly in the course of nephritis, in which case they are usually called uræmic, also in malaria and other diseases. Direct pressure from tumors of the brain or from hydrocephalus may in like manner cause convulsions of either a localized or a general form. Finally, we may have these nervous explosions in epilepsy, such as I have already described when speaking of that disease (page 724).

It will be well to remember that this entire class of central convulsions emanates from the brain; also that where the convulsions are unilateral or localized we should suspect a central rather than a peripheral origin.

The other class of convulsions, which I have spoken of as of peripheral origin, and which are called reflex, have so many causes that it would scarcely be advisable to attempt to enumerate them all. Convulsions of this class may arise from almost any source in infants whose nervous system is so easily irritated that the slightest cause may produce a nervous explosion.



The disease which most commonly gives rise to convulsions of the reflex form is rhachitis. Rhachitic children seem to be predisposed to spasmodic attacks of all kinds, and a general clonic convulsion in children with rhachitis corresponds to the spasmodic contractions in the larynx which occur in rhachitis, and which I have already spoken of as laryngospasmus.

It is probable that there is no especial lesion in connection with the rhachitis which necessarily gives rise to convulsions, but that all the tissues in this disease are especially sensitive to causes which may produce reflex explosions. The most common cause of reflex convulsions in infants is improper food. Convulsions from this cause arise not only where manifestly indigestible articles are given to young children, but even in infants who are being fed from the breast. In the early days and weeks of life, before the breast has acquired its normal functions connected with elaborating a milk in which the solids are in proper proportion to each other and to the water, it is not uncommon for the infant to have convulsions produced by a disturbance of the mammary function. In cases of this kind it is usually found that the percentage of the proteids is high, and that the convulsions will continue until this high percentage has been lessened, if the infant is allowed to continue to nurse. Whether the teeth of themselves during their development are a source of sufficient irritation to produce convulsions has been questioned by many observers. It is, however, evident that during the different periods of dentition reflex convulsions are more apt to occur than when a tooth is not disturbing the infant's nervous system. In addition to the convulsions arising from improper food in the stomach during the dental period, foreign bodies in the intestine, whether in the shape of food or in that of intestinal parasites, may cause reflex convulsions. Foreign bodies in the nose and in the ear have been known to produce convulsions, as also has an inflamed tonsil in the initial stage of a follicular tonsillitis. Hot baths are so often given to infants when they are in convulsions that they should be spoken of as a source of convulsions, for they have been known to produce this result when care has not been taken to test the temperature of the bath before the infant is put into it.

PROGNOSIS.—The prognosis of infantile convulsions must, as you will readily understand, vary much in connection with the especial cause. On recovering from the attack the infant may show signs of some serious central lesion, such as paralysis, or may be left apparently perfectly well. A single convulsion followed by perfect recovery is of slight consequence, but where the convulsive attacks recur frequently and last longer than in the attacks which I have just mentioned, the prognosis becomes graver. Even though no central lesion be present, continued convulsions may by the shock to the infant's vitality finally cause death from exhaustion, or death may occur from spasm of the glottis. We must, therefore, no matter what the cause or what the apparent result of a convulsion may be, always look upon it as a grave symptom and endeavor to prevent its recurrence.

TREATMENT.—When you are summoned to treat an infant who is in convulsions, you should first see that the bath, in which you usually find that it has been immersed, is not too hot, and should order the infant to be taken out of the bath before it becomes conscious, or it may be so frightened as to excite again the reflex spasm. You should quickly examine the thorax for pulmonary and cardiac lesions, and should make inquiries as to the history of the case, especially as to the infant's diet. The temperature should be taken, and you should notice whether the fontanelle is bulging or depressed.

Having obtained this information, you can eliminate quite a number of causes for the attack, such as the onset of one of the exanthemata if the temperature is normal, and reflex convulsions from food or foreign bodies in the nose or in the ear. You can soon determine whether the convulsions arise from exhaustion, so that you can proceed at once to order stimulants, if necessary, and, if the convulsions continue, to make use of the general treatment which is indicated for all forms of convulsions.

You should be prepared to act promptly, and for this reason you should acquire the knowledge which will enable you readily to classify the attack under its proper head and thus treat it intelligently. The parents are so terrified when a convulsion attacks an infant that it is necessary for the physician to be able to inform them as soon as possible whether or not the convulsion is injurious. In order to aid you in differentiating the various causes of convulsions from each other I have prepared this table (Table 106).

TABLE 106.

*Infantile Convulsions.*

Central.	Peripheral (Reflex).
I. Diseases with high temperature. (Insolation, meningitis, the exanthemata, pneumonia, and others.)	Rhachitis. Food. Intestinal parasites.
II. Diseases accompanied by vascular stasis. (Pertussis, cardiac disease, tumors, hydrocephalus.)	Dental irritation. Foreign bodies in the ear and nose. Hot baths.
III. Diseases characterized by anæmia and exhaustion. (Loss of blood, diarrhœa.)	Mental disturbance, such as fright, and numerous other causes.
IV. Various toxic causes, such as drugs, or uræmia. (Belladonna, nephritis.)	
V. Organic central lesions. (Cerebral paralysis, or any other lesion of the brain.)	
VI. Presumably organic disturbance of the brain. (Epilepsy.)	

The treatment of infantile convulsions should be directed to the especial cause of the convulsion. In general, however, as often when the convulsion is present it is impossible to determine whether it is of central or of periph-



eral origin, it becomes necessary to endeavor to control the attack at once. For this purpose in all forms of convulsions the inhalation of ether in small amounts, and the emptying of the bowels by means of enemata, are indicated. Where the convulsions are of a very severe type, continuing with perhaps intermissions of only a few minutes, and the infant's life is evidently in danger from the continuous shocks which are taking place in its nervous system, a rectal injection of 0.6 gramme (10 grains) of bromide of potassium and 0.3 gramme (5 grains) of hydrate of chloral in 30 c.c. (1 ounce) of warm water, repeated if necessary every hour for three or four doses, is indicated. If the convulsions still continue and a fatal issue is anticipated, a subcutaneous injection of sulphate of morphine, beginning with 0.001 ( $\frac{1}{60}$  grain), should be tried.

In most cases of infantile convulsions, of whatever form, the warm bath at a temperature of not over 37.7° C. (100° F.) can be used, for, although it is not in any sense curative, it tends to quiet the nervous excitability and to lessen the muscular strain produced by the continuous spasmodic muscular contractions. The class of cases in which this is contra-indicated are those which are caused by a loss of blood, an anæmic condition, diarrhœa, and cardiac disease, and those in which venous stasis exists with a lowered temperature. In these cases stimulants are indicated.

In those cases which are symptomatic of the diseases which I have already spoken of as accompanied by high temperature, the application of cold to the head and the administration of the bromides are indicated.

The treatment of convulsions caused by the other diseases which I have enumerated is simply symptomatic while the convulsions continue, and later the proper care of these diseases. All the reflex convulsions from various causes are treated in like manner symptomatically and by the removal of the especial cause.

I have already shown you an infant (Case 342) in clonic convulsions, and described to you the characteristics of the attack, while speaking of epilepsy (page 727).

I have here a few cases which may be of interest in this connection for you to see.

This little girl (Case 374) is six and one-half years old. She was healthy at birth, and has never had any disease. For the last three years she has from time to time had a convulsion, clonic in type. When in the convulsions she does not bite her tongue. The first convulsion occurred when she was three years old; the next when she was four years old; the next when she was four and one-half years old; and the last one when she was five years old.

As all these convulsions have apparently been produced by the same cause, it will only be necessary to describe them in a general way. They have been characterized by sometimes continuing much longer than is usual in infantile convulsions, one of them having lasted for one hour and a half, during which time the hands were clinched, the eyes were rolled up, and the entire body and limbs were convulsed. Previous to each attack the child for a number of days has had indefinite symptoms which she could not describe accurately, connected with the abdomen and accompanied by a feeling of weakness and slight muscular twitching.

At the time of the earlier attacks her mother found that these symptoms could be dissipated and apparently a convulsion prevented from occurring by giving her a dose of castor oil about once a week. After the third convulsion she passed a lumbricoid worm. From that time whenever she showed the premonitory symptoms of a convulsion she was treated with santonin followed by a cathartic, a lumbricoid worm was each time passed, and the symptoms disappeared. When she was five years old she was thoroughly treated for these lumbricoid worms with santonin, and after a large ascaris had been passed the nervous symptoms ceased entirely. The child has now not had a convulsion for over a year. There has been no reappearance of the worms.

This child represents the class of cases which I have described when speaking of reflex convulsions, the cause of the peripheral irritation evidently being an intestinal parasite.

I have here an infant (Case 375), thirteen months old, whose nervous system has always been in so irritable a condition that the slightest cause was sufficient to produce a convulsion.

When he was eight months old he had an attack of pertussis, and during the course of the disease he had a number of convulsions. At one time when the pertussis was at its height he had from fifteen to sixteen convulsions within thirty-six hours, each of them lasting from five to ten minutes.

When the first teeth began to press upon the gums he occasionally had a convulsion. In addition to the general muscular spasms he had nystagmus of the right eye. For the last two or three months he has had no convulsions, and the nystagmus is much less noticeable.

This boy (Case 376), four years old, has from time to time had convulsions, which, so far as I can ascertain, are simply reflex, and are not connected with epilepsy or with any organic disease.

When he was six months old he had a number of convulsions while cutting his incisors. When he was two years old he had an attack of epidemic influenza, which was ushered in by a convulsion; and the same phenomenon occurred when he had an attack of catarrhal laryngitis some months later.

This little girl (Case 377), four years old, is, as you see, a bright child, and is in fairly good health. She is apparently recovering from convulsive attacks which occurred with great frequency in her second and third years, and which were apparently produced by epilepsy. At one time she had fifty-four convulsions in forty-eight hours.

She has been treated simply by carefully regulating her diet and with bromide of potassium.

The prognosis in this case is not very favorable, as she is probably an epileptic, and the convulsions are liable to return at any time, especially as puberty is approached.

This next child (Case 378), a girl, four years old, is a case of considerable interest, as presenting an instance of some slight organic lesion occurring when she was two and one-half years old, accompanied by a convulsion. The convulsion was of the general clonic type, lasted for a few minutes, and was accompanied by a temperature of  $39.7^{\circ}\text{C}$ . ( $103.5^{\circ}\text{F}$ .) and a pulse of 140. On recovering from the convulsion she was found to have a slight hemiplegia of the left side, which lasted for only a few hours. She then recovered entirely, and has since had no convulsions, but she has never developed either mentally or physically in accordance with what would be expected in a child of her age, so that she has to be watched over by her nurse as carefully as though she were three years old, as she is liable to fall and does not go up-stairs easily.

This child (Case 379), three and a half years old, has always been well and strong. She was suddenly attacked about a month ago by a chill, and was found to have a high temperature and a quick pulse. A few hours later, the temperature having risen to  $40^{\circ}\text{C}$ . ( $106^{\circ}\text{F}$ .) and the pulse to 170, she suddenly had a general clonic convulsion. After the convulsion had ceased she remained unconscious, and some hours later had another convulsion. She was placed in a warm bath, and after the temperature had been reduced two or three degrees the convulsive movements ceased. A little later a general papular efflorescence of measles appeared on her face and neck, afterwards spreading to the body and limbs. She



became perfectly conscious, and did not have any other severe symptoms during her attack of measles, nor any return of the convulsions.

Her case is an instance of convulsions produced by a high temperature in the initial stage of one of the exanthemata.

The next three infants whom I have had brought here to show you are interesting examples of the necessity of regulating the solid constituents of the milk which is given to young infants.

The first case (Case 380) is that of a little girl, four months old. Her mother, who was strong and well and apparently had plenty of good breast-milk, nursed her at birth. When she was three months old she began to have convulsions, which occurred almost every hour. Suspecting that the proportion of solids in the breast-milk was too high for the infant to digest them, and that they were producing a peripheral irritation which was the cause of the reflex convulsions, I had an analysis of the milk made, and found that the proteids showed a percentage of from 4 to 5. The infant was then fed with a carefully modified milk in which the percentage of the proteids was made 1. Within a few hours the convulsions ceased, and they have never returned. As you see, the infant is perfectly well and thriving to-day.

I have in instances of this kind so regulated the percentage of proteids in the mother's milk by the means which I have described to you in a previous lecture (Lecture VII., page 188) that an infant who before this modification of the mother's milk had been made was having continued convulsions ceased entirely to have them, and was nursed successfully for many months.

This next infant (Case 381), a little girl, six weeks old, began to have convulsions when she was four weeks old. The convulsions occurred every twenty minutes for twenty-four hours, and sometimes as often as every fifteen minutes. They lasted for only a few seconds. The infant was being fed on the milk of a Jersey cow. She was then fed on a carefully modified milk with a moderate percentage of fat and proteids, and the convulsions did not return.

The third case (Case 382) is a boy, six weeks old. He was healthy and strong at birth, and was nursed by his mother for three weeks. During this time he gained in weight and digested the milk perfectly. The mother, however, was unable to continue nursing him after the third week, and it was decided to feed the infant on modified milk. The prescription for this modified milk sent to the laboratory by a physician was as follows:

PRESCRIPTION 72.

Fat . . . . .	5.50
Sugar . . . . .	7.00
Proteids . . . . .	3.50

Soon after this milk was given to the infant it began to have convulsions, which continued for twenty-four hours, at intervals of two or three hours, until the milk was omitted. Another modification of the milk was then substituted for the first, and the infant ceased to have convulsions and has since digested the milk perfectly. The percentages in the last prescription were as follows:

PRESCRIPTION 73.

Fat . . . . .	3.50
Sugar . . . . .	6.50
Proteids . . . . .	1.50

**TREMOR.**—Universal or partial tremor is, in my opinion, rare in infancy and early childhood in comparison with later life. It does, however, occur, and is usually significant of an organic cerebral lesion. I have noticed it also in cases of infantile atrophy, where as recovery gradually took place the tremor disappeared. In this form it appears to be chiefly a symptom of weakness. It may be quite marked as a general symptom, but it is not especially significant as connected either with the diagnosis or with the prognosis.



## LECTURE XXXIX.

## THE MYOPATHIES.

PROGRESSIVE MUSCULAR ATROPHY.—PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.—  
MYOTONIA CONGENITA (THOMSEN'S DISEASE).

**PROGRESSIVE MUSCULAR ATROPHY.**—Progressive muscular atrophy is a name used to denote certain conditions which were originally supposed to be due to a disease of the spinal cord. Later, however, it was found that two forms of lesions produce this atrophic condition of the muscles. One of these, the neuropathic form, is an affection of the spinal cord, and is designated the Aran-Duchenne or thenar type. The other form is found to be a primary disease of the muscles, and is classed as one of the myopathies.

**NEUROPATHIC PROGRESSIVE MUSCULAR ATROPHY.**—The neuropathic atrophies are so rare in infancy and early childhood that little need be said concerning them. The neuropathic progressive muscular atrophy is caused by a chronic degeneration of the ganglion-cells of the anterior cornua, and this is the form which I have just alluded to as the Aran-Duchenne type. In this form the atrophy almost always begins in certain muscles of the hand, especially those of the ball of the thumb, and it is for this reason called thenar. It is rarely seen before the twentieth year, and is not hereditary. Hypertrophy of the muscles does not occur.

**MYOPATHIC PROGRESSIVE MUSCULAR ATROPHY.**—The myopathic atrophies show a marked hereditary tendency. They have been divided by various authors according to the different portions of the body in which they begin. The disease in each case is essentially the same, and this division seems to be unnecessary and misleading, because, although the affection may begin in any part of the body or extremities, yet, as a rule, it may be said that the primary myopathies begin in the muscles of the shoulder, face, or back. In all these cases the atrophy usually begins before the twentieth year.

Where the muscles of the face and scapulo-humeral groups are involved early, it is called the *facio-scapulo-humeral type* of Landouzy and Déjerine.

Where the atrophy begins in the gluteal muscles and those of the thigh, arm, and shoulder, it is called the *juvenile type* of Erb.

Where the atrophy first affects the muscles of the legs, it is called the *peroneal type*, and the affection shows itself in the peripheral muscles of the lower extremities, such as the extensors of the great toe, and afterwards in the common extensor of the toe and in the peroneal group. There is, however, regarding this latter type a doubt as to whether it is a primary myopathy.

**PATHOLOGY.**—According to Delafield and Prudden, the lesion of progressive muscular atrophy consists essentially in a combination of simple or degenerative atrophy of the muscular fibres with chronic interstitial inflammation, and is sometimes associated with proliferative changes in the nuclei of the muscles. In the earlier stages of the disease the muscles may be pale and soft, but exhibit macroscopically little alteration. Gradually, however, the muscular substance is replaced by connective tissue, so that in marked and advanced cases the muscles are converted into fibrous bands or cords, the cicatricial contractions of which may induce great deformities.

Microscopic examination in the early stages of the disease shows a proliferation of cells in the interstitial tissue, so that this may have the appearance of granulation or embryonal tissue; also in some cases marked proliferative changes occur in the nuclei of the muscles, leading to the formation of new cells, which may more or less replace the contractile substance within the sarcolemma. The new interstitial tissue increases in quantity and grows denser, and may crowd the muscular fibres apart. The walls of the blood-vessels may also become thickened. Accompanying these interstitial alterations the atrophy of the fibres of the muscle proceeds. These may simply grow narrower, retaining their striations, or they may split up into longitudinal fibrillæ or transversely into discoid masses, and in this condition disappear. In other cases a certain amount of fatty or hyaline degeneration may be present. These degenerative and proliferative changes do not, as a rule, occur uniformly in the affected muscles, but some parts are affected earlier and more markedly than others. The atrophied muscles may be replaced by fat. The atrophy is essentially chronic, affecting the different fibres gradually and not the whole muscle at once.

**SYMPTOMS.**—The symptoms of myopathic progressive muscular atrophy are those connected with a wasting of the muscles.

In the *facio-scapulo-humeral type* the atrophy of the muscles begins at an early age, and usually involves the face first. Both sides of the face are commonly affected, although the disease may be unilateral for a long period. The muscles chiefly affected are the orbicularis oris, the zygomaticus, the orbicularis palpebrarum, the frontalis, and the buccinator. The levator anguli oris may also be affected, but usually is not. In connection with this progressive atrophy of the face the muscles of the shoulder and upper arm are often affected. Landouzy and Déjerine have reported an autopsy in a case of this facial variety in which the lesions were a primary degeneration of the muscles and a very slight increase of connective tissue and fat. In this connection I would mention that a form of what is called facial hemiatrophy without the involvement of any other muscles occurs between the fifth and twelfth years of life.

In the "*juvenile form of Erb*" the muscles affected are usually the pectoralis minor and pectoralis major, the trapezius, the rhomboideus minor and rhomboideus major, the latissimus dorsi, the whole group of spinal extensors, the triceps, the brachialis anticus, and the biceps.



In all these forms the muscles react to both the faradic and the galvanic current, and there is no reaction of degeneration.

**DIAGNOSIS.**—The diagnosis of myopathic progressive muscular atrophy should first be made from the neuropathic form. The former is hereditary; the latter is not. In the former the atrophy usually begins in the muscles of the shoulder, face, and back, while in the latter it is exceedingly rare for it to begin elsewhere than in the muscles of the hand. Hypertrophy of certain muscles and the beginning of the atrophy early in life, usually before the tenth year, are characteristic of the myopathic variety, in contradistinction to the late development and the absence of hypertrophy in the neuropathic form.

The muscular atrophy which accompanies certain cases of chronic multiple neuritis may be mistaken for a myopathic affection, and must therefore also be differentiated. At times the resemblance of the two diseases is quite striking, but it does not last for a sufficiently long time to leave the diagnosis in much doubt. You must remember that chronic multiple neuritis is never hereditary, that the paralysis which accompanies it is out of proportion to

## CASE 383.

I.



II.



The facio-scapulo-humeral type of primary myopathic atrophy. I. Before the disease began.  
II. After the disease was well advanced.

the atrophy, and that there may also be distinct symptoms of ataxia, all of which symptoms are unusual in the primary myopathic atrophy.

**PROGNOSIS AND TREATMENT.**—The prognosis is very unfavorable, and there is no known treatment which benefits the disease. The patient should be placed under the most favorable surroundings for his general health.

Precautions should always be taken to prevent the contractures which necessarily occur in the later stages of the disease from producing awkward positions of the body and limbs.

The *facio-scapulo-humeral type* of primary myopathic atrophy is so exceedingly rare that I am fortunate in having a case here to-day to show you. It has been carefully attended in my wards by my colleague Dr. Bullard.

## CASE 383.

## III.



The facio-scapulo-humeral type of primary myopathic atrophy. Female, 10 years old.

This little girl (Case 383), ten years old, is of healthy parentage. There are four other children in the family, who show no signs of disease. This child, although she has had various diseases, such as variella, measles, and pertussis, has on the whole been well and strong, and until three years ago was unusually well developed. Here is a picture (I., page 765) of her taken at that time, just before she was attacked with the disease from which she is now suffering.

You see that the face is unusually full and plump, and at that time there was evidently no sign of muscular disturbance.

If you will now look at the child's face as she stands before you (II., page 765) and compare its emaciated old look with the young, well-nourished look shown in the picture (I.), you will at once understand that she is affected by a disease of serious import.



Three years ago she had an attack of epidemic influenza. Since then she has been losing in weight and strength. She has complained of pain in the abdomen, not localized, but dull, continuous for a few hours, and then remitting for an hour or so. This disturbance will last for two or three days, and during this time she does not care to do anything, but lies down, usually on her back. She occasionally vomits; there is nothing characteristic about the vomiting, but it relieves the pain of the acute attacks. She may have at times slight nausea; she seldom has headache; the bowels move regularly; she has no cough, but a slight nasal catarrh is usually present, as she catches cold very easily. She also has enuresis, and during the acute attacks of pain she is apt to have attacks of pavor nocturnus. She is very nervous, and cries easily.

## CASE 383.

## IV.



The facio-scapulo-humeral type of primary myopathic atrophy.

On examining the child in front as she sits on a stool (III., page 766) you see that the legs are unusually well developed, in marked contrast to the atrophy of the face, body, and arms. Her respiration is free and equal on both sides. The face and neck are extremely thin, and the muscles are atrophied. The muscles of the upper extremities and chest are thin and small, but firm and of fair strength, while those of the abdomen and legs are well nourished and firm. The skin is dry, the eyes are bright, and the reaction of the pupils is normal. On physical examination nothing else abnormal is found, with the exception that

the action of the heart is rather rapid. There is slight ankle-clonus, and the patellar reflexes are slightly increased. The glands of the neck, axillæ, and groins are very slightly enlarged. The tongue is normal and can be protruded steadily. The examination of the urine shows it to be normal, with the exception of a slight trace of albumin.

The only other case of this disease which has to my knowledge been reported in this country is one by Osler.

On examining the child's muscles more closely you will see (III.) that the most marked atrophy is situated above the diaphragm, while the abdomen and legs are remarkably well developed.

The muscles affected are those which I have already enumerated in the general description of the disease.

On turning the child so as to look at her back (IV., page 767) and making her stand up, you will notice the striking difference between the arms and upper part of the body and the legs and lower part.

**PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.**—The form of primary muscular atrophy which I shall next speak of is what is called pseudo-hypertrophic muscular paralysis. This disease is characterized by a diminution of power in certain muscles, accompanied by an abnormal increase in their size and a diminution in the size of other groups of muscles. Although an apparent increase in the size of the muscles takes place, yet the enlargement is produced by an hypertrophy of the connective tissue and an unusual deposit of fat.

The disease affects males more frequently than females. It usually occurs between the ages of two and eight, although in exceptional instances its appearance is delayed until about the twentieth year.

**PATHOLOGY.**—According to Gowers, the pathology is represented by a primary interstitial change in the muscles, showing a growth of fibrous tissue or of fat-cells which produces an increase in the size of the muscles. The muscular fibres are secondarily affected by this interstitial change, and are apparently narrowed by pressure. Atrophy, which is an especial feature of the affection, exists in the later stages of the disease in the muscles of the legs, and is frequently the primary pathological change in the muscles of the trunk and upper extremities. The development of fat-tissue between the atrophied fibres may prevent any diminution in the apparent size of the muscles. This often, in the muscles of the calves, may cause them to be much increased in size. In the upper extremities the deltoid and triceps are most commonly involved, in the lower the gastrocnemii. The infra-spinatus, the latissimus dorsi, and the pectoralis major muscles are also commonly affected. The lesion is usually symmetrical, affecting similar muscles on both sides of the body, but it may be unilateral. The muscles may be affected partially or completely.

**SYMPTOMS.**—The disease develops slowly, and the symptoms are those which would naturally be expected from the muscular lesions, and are quite characteristic.

The first symptoms usually noticed are a weakness of the muscles, and a shuffling, unsteady, swaying gait, with the feet apart and a tendency to stumble and to fall. The children get tired very easily, and are noticed



to have difficulty in walking up-stairs. These general symptoms usually precede any noticeable enlargement of the muscles. The position on standing is apt to be that of lordosis, and on sitting down this curvature disappears.

The next symptom which is noticed is an enlargement of the calf-muscles, which are usually hard and firm. In addition to this there is an atrophic condition of the muscles of the shoulders and back. The muscles next to those of the calves which are most likely to be affected are the extensors of the leg, the glutei, the lumbar muscles, the deltoid, triceps, and infra-spinatus. The muscles of the neck, face, and upper arm are usually not affected, but in rare cases these muscles, as well as those of the tongue, have been involved. Exceptionally an hypertrophy begins in the upper extremities, and in these cases the deltoid muscle is usually first affected. At times only part of the muscle is involved. These children learn to walk late, and assist themselves by leaning on the furniture or other objects in their path. Sometimes when they are kneeling on the hands and knees there is noticed a very characteristic saddle-shaped depression of the back, which is due to the weakness of the erector spinæ muscles. This, however, is a symptom of a late stage of the disease. When the child is placed on the floor on its back it has difficulty in getting up. It has to turn over on its face first, and then to aid the weakened muscles of the legs and trunk by means of the hands and arms, climbing up, as it were, upon itself by placing the hands upon the knees and then farther and farther up the thighs. Fibrillary contractions do not occur. The knee-jerks in some cases disappear as the disease advances. Sensation, as a rule, is normal. There is seldom any disturbance of the bladder or rectum.

As the disease advances, the pseudo-hypertrophic condition disappears and is succeeded by atrophy. In some cases the atrophy occurs without the preceding hypertrophy. In the later stages of the disease contractions of the muscles occur, and in this way permanent distortions of the joints may result. The most common deformities are talipes equinus and flexion of the knees and hips. There may be such a contraction of the biceps as to prevent full extension of the arm, and in some cases the contraction of the muscles of the calves is so great as to prevent the child from placing the heels upon the ground.

According to Bradford and Lovett, lateral curvature of the spine may occur, and at other times a permanent flexion of the spine from weakness of the erector spinæ muscles, so that the child sits bowed forward. The electrical reactions are not altered to any degree until the muscles have reached a marked stage of atrophy, then they are diminished in proportion to the muscular wasting, and finally they are lost. The reaction of degeneration is never present.

DIAGNOSIS.—The diagnosis of pseudo-hypertrophic muscular paralysis when the disease is well established is not difficult. The peculiar gait, the size of the calf-muscles, entirely out of proportion to their strength, and the characteristic manner in which the child rises from the floor, at once sug-

gest this affection. Gowers also attaches diagnostic importance to the co-existence of enlargement of the infra-spinatus and wasting of the latissimus dorsi and the lower part of the pectoralis major muscles. We should remember that in pseudo-hypertrophic muscular paralysis, in contradistinction to progressive muscular atrophy, the small muscles of the hand and of the face are, as a rule, not affected, that pain is usually not present, and that changes in the nutrition of the skin and nails do not occur.

## CASE 384.

## I.



Pseudo-hypertrophic muscular paralysis, showing enlarged calves.

We differentiate true muscular hypertrophy from pseudo-hypertrophic paralysis by the strength which accompanies the former, and by the weakness which occurs in the latter.

In the early stages of the disease it is at times difficult to distinguish simple backwardness in walking from early pseudo-hypertrophy, but the characteristic symptoms of pseudo-hypertrophy, which develop very soon, do not leave the diagnosis long in doubt. The same thing may be said of differentiating pseudo-hypertrophy from the muscular disturbances occurring in idiocy, spastic paralysis, rhachitis, and Pott's disease.



**PROGNOSIS.**—Recovery in this disease is unknown, and the children seldom live to middle life. Death usually occurs from some intercurrent disease. The course of the disease is chronic. The muscular weakness, the lordosis, and the peculiar gait last for several months or a year. The hypertrophy of the muscles then begins, and continues progressively for one or two years, when it reaches its maximum and becomes stationary, remaining so for several years or even longer. A stage of increasing feebleness and extension of the paralysis then begins, the muscles become more wasted, and the power of motion is lost in the legs and arms. Sometimes the disease after remaining stationary rapidly advances to a fatal issue.

**TREATMENT.**—At present we know of no way of curing the disease. Massage has proved to be more beneficial than the use of electricity in these cases. Systematic muscular exercise, for the purpose of preserving the nutrition of the unaffected muscular fibres and to ward off the permanent contractures, is indicated. Where the muscles are drawn up, tenotomy is often of much use, and division of the tendo Achillis on both sides may for a long time restore the power of walking. Bradford and Lovett advise tenotomy of the hamstring tendons also, in severe cases. Strict attention to the health and hygiene of the patients, combined with muscular exercise and tenotomy, will often improve the general condition for a considerable period of time.

## CASE 384.

## II.



Pseudo-hypertrophic muscular paralysis. Showing position assumed in rising from the floor.

I have here a case which represents certain points which I have just spoken of in describing pseudo-hypertrophic muscular paralysis, and which I am enabled to show you through the kindness of Dr. Rupert Norton.

Looking at this boy from behind (Case 384, I., page 770), you will notice at once that the calves of the legs are greatly enlarged.

On making the boy lie down on the floor and then telling him to get up (II., page 771), you will notice that he assists himself by putting his hands on his knees and gradually higher and higher on the thighs until he assumes the erect position.

I shall now ask you to look at these illustrations of two brothers which I am enabled to show you through the kindness of Dr. H. N. Thomas, of Baltimore.

The history of these cases (Cases 385 and 386) is as follows. The smaller boy is eight years old, the larger ten years old. They have always lived in the country, and no mention of any especial disease has been obtained, but the history of both cases is unsatisfactory.

#### CASES 385 AND 386.

I.



II.



Pseudo-hypertrophic muscular paralysis. Brothers, 8 and 10 years old. I. Showing atrophy of back and enlarged calves. II. Showing the lordosis.

The older boy began to walk when he was nineteen months old, but was clumsy and never walked well. When he was seven years old he began to have difficulty in going upstairs, and it was noticed that the calves of his legs were growing larger, while his arms were becoming smaller. The curve in his back was first noticed when he was eight years old. When nine years old he lost the power of walking, and is said to have grown rather stupid.



The younger boy was always delicate, but never had any disease, and began to walk when he was fifteen months old. He learned to walk pretty well, but when he was four years old he began to show weakness in the legs and ankles, and this weakness increased steadily. When he was six years old his arms began to get smaller and his legs to increase in size.

You see that both boys show marked lordosis.

**MYOTONIA CONGENITA** (Thomsen's Disease).—A third form of primary myopathy, which is usually termed Thomsen's disease, from the name of the physician who first thoroughly described it, is characterized by an inhibition of the voluntary movements. This disturbance of movement is due to a stiffness and tension of the muscles occurring at the commencement of motion. The most important etiological factor in the disease is that it is hereditary. In almost every case it begins in early childhood.

The pathology of the disease has not been accurately established by means of autopsies, but an examination of sections of muscle taken from these cases has shown, according to Erb and Jacoby, that the morbid changes are the result of an enormous hypertrophy of all the muscular fibres, great proliferation of the nuclei, and a slight increase of the perimysium. The disease appears to be a congenital affection of the muscular fibres.

The symptoms of this disease are noticed only during voluntary movements, the contraction of the muscles responding very slowly to the will, and persisting for a little time after the individual has willed the muscular movement to cease. The muscles of the arms and legs are those usually implicated. The sensation and reflexes are normal. The muscles are apparently enlarged, giving at times the appearance of hypertrophy, but the strength of the muscle is not proportionate to its size. Erb has described a characteristic electrical reaction, called the myotonic reaction, in which the contractions caused by either current attain their maximum slowly and relax slowly, and wave-like contractions pass from the cathode to the anode. One of the peculiarities of the disease is that when exertion is made, such as attempting to go up-stairs, the muscles which previously were quiescent become very stiff and will scarcely respond to the will. Another peculiarity is that long-continued rest makes the disorder worse. It is also exaggerated by heat, cold, and excitement.

Since the discovery of the myotonic reaction the diagnosis of the disease is not difficult.

Although at times it may recover temporarily, the disease is incurable, and there is no known treatment which is of benefit to it.

## DIVISION XII.

### DISEASES OF THE MOUTH, NOSE, NASO-PHARYNX, AND PHARYNX. DIPHTHERIA.

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#### LECTURE XL.

##### DISEASES OF THE MOUTH.

STOMATITIS CATARRHALIS.—STOMATITIS HERPETICA.—STOMATITIS ULCEROSA.—STOMATITIS MYCETOGENETICA.—GLOSSITIS.—MICROGLOSSIA.—MACROGLOSSIA.—DIFFICULT DENTITION.

IN speaking of diseases of the mouth you must understand that much confusion exists as to the nomenclature of this class of affections. Thus, a great variety of names has been used by different authors to describe the same disease, so that at times it is quite difficult for one investigator to compare his work with that of another. Such terms as “canker” and “aphthæ” are so commonly used for almost any morbid condition of the mucous membrane of the mouth that they have ceased to convey any definite idea.

It has therefore been found necessary, in order that any advance should be made in this most difficult department of medicine, to adopt some more exact nomenclature, so that physicians in different parts of the world should in every case use the same name for the same disease. In furtherance of this object the American Pediatric Society has adopted a provisional nomenclature of the diseases of the mouth which was prepared by Dr. Forchheimer, of Cincinnati, and myself. To Dr. Forchheimer's extended investigations on this subject I am much indebted.

Diseases of the mouth occur more frequently and in much greater variety in infancy and in early childhood than at any later period of life. This depends partly on the anatomical conditions at different periods of development and partly on the external influences which are brought to bear upon the buccal mucous membrane. During the first three or four months of life the function of the salivary glands is not developed, and the flow of saliva is insignificant. This lack of saliva allows the mucous



membrane of the mouth to be dry in comparison with that of the older subject. Even after the saliva is secreted the infant is more apt under certain conditions to let it flow from the mouth than to swallow it, so that the mucous membrane of the lips and mouth may present a different appearance in young infants, when they are attacked by various morbid processes, from that seen at a later period of development. We must also remember that the salivary glands in addition to their especial function are excretory organs, and that substances which are absorbed by the stomach may be eliminated by the mouth and in this way become sources of irritation and disease in the latter. The mucous membrane of the mouth during almost the whole period of infancy is subject to external sources of irritation to which older children, as a rule, are not liable. Thus, during the first year the mucous membrane is subjected to more or less mechanical irritation through the mechanism of sucking. At this period, also, it is very common for foreign organisms to be introduced into the mouth by means of the fingers either of the infant itself or of its attendant. It is not surprising, therefore, that we should meet with a great variety of pathological conditions in the mouth in infancy.

The organisms which occur in the mouth are so numerous that very few of them have as yet been differentiated in such a way that they can be known as the cause of the specific disease in which they are often found. We cannot, therefore, at the present time describe the various diseases of the mouth under their proper etiological headings, and we are forced to adopt provisionally the name of the pathological lesion which exists in them.

In almost every disease of the mouth which occurs in infants and in young children you will find a coexisting inflammation of the mucous membrane of the mouth. This inflammation may at times be very mild and often difficult to detect as such, but it still presents a recognizable pathological condition. This inflammatory condition, though not necessarily preceding the various diseases, yet in a large number of cases either exists as a basis on which the disease develops, or so closely accompanies it that the general name *stomatitis* (inflammation of the mucous membrane of the mouth) seems to be a proper term to use in connection with all these diseases.

Under the general heading *stomatitis* we can speak of most of the important diseases which affect the mucous membrane of the mouth in infancy and early childhood. These diseases may be divided into four general headings, according to the character of the lesions which occur in them. In order that you may readily understand the classification of each disease when I speak of it, I shall first show you a table (Table 107) of the provisional nomenclature which has been adopted by the American Pediatric Society.

TABLE 107.

*Provisional Nomenclature of Diseases of the Mouth.*

STOMATITIS . . .	{	Catarrhalis . . . . .	{	Simplex.	
			{	Exanthematica . . . . .	{ Secondary to the Exanthemata.
				Traumatica . . . . .	{ Mechanical.
					{ Thermal.
					{ Chemical.
		Herpetica . . . . .		Aphthosa.	
	{	Ulcerosa . . . . .	{	Scorbutus.	
			{	Mineral Poisons . . . . .	{ Arsenic.
				And other diseases.	{ Lead.
					{ Mercury.
	{	Mycetogenetica . . .	{	Hyphomycetica . . . . .	Thrush.
			{	Pseudo-Membranosa . . .	{ Diphtheria.
					{ Tuberculosis.
					{ Syphilis,
					{ and like diseases.
			{	Gangrænosa . . . . .	Noma.

Following this table, you will see that the four general names which cover all these diseases are *stomatitis catarrhalis*, *stomatitis herpetica*, *stomatitis ulcerosa*, and *stomatitis mycetogenetica*.

**STOMATITIS CATARRHALIS.**—The form of stomatitis which is called the *simple* or *erythematous* form (*stomatitis simplex*) is commonly seen in young infants as a hyperæmic condition of the blood-vessels, causing diffuse redness of the whole buccal mucous membrane. This erythematous form is so common and so entirely without clinical significance that it may be considered as physiological and need only be referred to.

The second form, which is called *exanthematica*, is the condition of the mucous membrane which occurs secondarily to the exanthemata. This condition of the mucous membrane has already been described in connection with these diseases, and therefore need not be spoken of again.

The third form, which is called *traumatica*, is the one which represents the characteristic *stomatitis catarrhalis*. The causes of the traumatic form of stomatitis catarrhalis are very numerous. They may be *mechanical*, *thermal*, or *chemical*. Among the most common mechanical causes may be cited the irritation produced by rubber nipples, too vigorous cleansing of the mouth, injudicious rubbing of the gums during dentition, and local irritation from a tooth. The thermal form of traumatism may result from the administration of food which is too hot. The chemical irritation may arise in various ways, as from lack of cleanliness in the mouth, with its resulting fermentation, and from the elimination of irritating products from the glands apparently connected in some way with disturbance in the gastro-enteric tract. It is probable also that various forms of bacteria or their



products may cause both mechanical and chemical irritation of the buccal mucous membrane. Our knowledge of the bacteriology of the mouth is as yet, however, so limited that we can scarcely undertake to describe the relation between special forms of bacteria and special lesions of the mucous membrane.

**PATHOLOGY AND SYMPTOMATOLOGY.**—As the lesions which are seen in the mouth of an infant with stomatitis catarrhalis during life almost entirely disappear at death, and as very few post-mortem examinations have been made of these lesions, we can speak of the pathology and symptoms of this disease together.

The lesion is essentially an inflammatory one, and occurs in different grades. On examining the mucous membrane in these cases it is seen that the entire lining of the mouth is intensely reddened, that the temperature of the mouth is increased, that there is usually a certain amount of swelling, and that, although the mucous membrane may be under certain circumstances, especially at first, dry, yet, as a rule, later there is a hypersecretion of mucus and saliva. The blood-vessels are so distended and their walls are apparently so weak that the slightest traumatism may cause their rupture, and the saliva is frequently mixed with a little blood. In older children the mucous membrane may be considerably swollen, especially behind the incisor teeth. In addition to this general condition of the mucous membrane of the mouth, at times the lips are found to be swollen and much reddened. The surface of the mucous membrane shows a number of small round prominences, which are the muciparous follicles. If complete occlusion of the ducts of these follicles occurs, great dilatation of the gland will take place, and a cyst may be formed. This, however, is a comparatively rare complication. In connection with the disturbance of the glands in the mouth the lymphatic glands are usually involved secondarily.

When the catarrhal condition is at its height the mucous membrane is so vulnerable that even slight traumatisms may cause abrasions. The most marked symptom of stomatitis is pain. The infant is restless, usually has a heightened temperature, and refuses to take its nourishment. The saliva is acid in its reaction, and when secreted in large quantities flows out of the mouth upon the chin and neck, sometimes causing considerable irritation. The tongue is dry and white at first, then becomes of a grayish color, and as the secretion of saliva increases the coating of the tongue is washed off and its surface becomes red.

**PROGNOSIS.**—The prognosis of stomatitis catarrhalis is, as a rule, good. Although the disease does not run a definite course, yet in most cases after a few days the pathological condition improves and the symptoms grow less severe. The course of the disease is, however, often lengthened by the secondary conditions which arise from the gastric disturbances, which may be caused by swallowing the irritating secretions of the mouth. In weak, poorly nourished infants who refuse to nurse or to take the food which is given them, serious results may arise from a lack of sufficient nourishment,

so that in these cases the prognosis is always grave. In older children the disease may be considered to be of a benign nature.

**TREATMENT.**—Although stomatitis catarrhalis may run a favorable course without any treatment whatever, yet there are so many causes which may prolong its course or give rise to secondary affections that it is exceedingly important to treat the disease at once. The indications for treatment are to relieve the pain and to allay the irritation of the mucous membrane so that a sufficient amount of nourishment may be taken by the infant to prevent it from being harmed by a lack of nourishment or by a secondary disturbance of the gastro-enteric tract. If the cause can be ascertained, it should be removed at once. The local application of a one to two per cent. cold solution of bicarbonate or borate of sodium in distilled water is indicated. This solution should be used very gently every half-hour when the infant is awake, by means of a dropper, and occasionally on a clean swab of absorbent cotton. The infant should be systematically fed at regular intervals, whether it resists or not; and if it is not being nursed or will not suck from the nipple, a carefully modified milk at a temperature of about 32.2° C. (90° F.) should be administered with a spoon or dropper. There is no necessity for giving any drug internally in this disease.

Where the stomatitis proves to be intractable and lasts for more than three or four days, the mouth can be gently touched with a cotton swab wet with a one per cent. solution of nitrate of silver. This should be done once a day, and the mouth washed carefully with cold sterilized water after the application.

Where there are any abrasions which show a tendency to extend or to form an ulcer, they should be touched with a little nitrate of silver melted on the end of a silver probe. These abrasions are often so painful that in themselves they may prevent the child from taking its food, and after they have been treated with the nitrate of silver the child will often again take its nourishment readily.

I have here an infant (Case 387), six months old, who is a marked case of stomatitis catarrhalis. This infant is reported to have always been healthy, and is being nursed by its mother. It cut its first tooth, a middle lower incisor, when it was five months old. Nothing abnormal was noticed about the infant until two weeks ago, when it became fretful, restless, had a heightened temperature of about 38.8° C. (102° F.), and vomited occasionally. Although it did not cry a great deal, it frequently whimpered, as though in pain, and kept putting its fingers to its mouth. A few days later it refused to nurse. When it was put to the breast it appeared to be hungry and would take hold of the nipple vigorously, but immediately afterwards would draw its head away, as though sucking the nipple caused pain.

A physical examination shows nothing abnormal about the infant except in its mouth. The mucous membrane of the mouth, tongue, and gums is reddened, and small raised spots are seen corresponding to the positions of the muciparous glands. The mucous membrane of the tongue and lips is somewhat swollen and hot, and evidently sensitive to the touch. Where the tooth touches the tongue the inflammatory condition is especially marked, and it is possible that the sharp edge of the tooth was the original starting-point of the general inflammation which is now present.

In this case the indications are for active treatment, as the infant is losing in weight



from lack of sufficient nourishment, and if this continues the prognosis will soon become grave. When the mouth is in this condition there is also a great liability to other diseases being implanted upon it, as the mucous membrane is very vulnerable when a pronounced stomatitis catarrhalis is present. The saliva is flowing from the mouth in such quantities and is so irritating that an eczematous condition has been produced by it on the chin. The child is rather apathetic and does not like to be disturbed. The treatment which I shall order in this case is that the mouth be carefully washed with the following solution (Prescription 74):

PRESCRIPTION 74.			
<i>Metric.</i>		<i>Apothecary.</i>	
	<i>Gramma.</i>		
R Sodii boratis . . . . .	1   8	R Sodii boratis . . . . .	gr. xxx;
Glycerini . . . . .	7   5	Glycerini . . . . .	ʒii;
Aq. destil. . . . .	ad 120   00	Aq. destil. . . . .	ad ʒiv.
M.		M.	

This should be applied every hour while the child is awake. The chin should be frequently dried gently and a little vaseline applied to the eczematous surface. Until the child is willing to nurse again, the milk should be given by means of a dropper regularly every two hours. Under this treatment I shall expect rapid improvement within four or five days.

**STOMATITIS HERPETICA.**—The name herpetica has been adopted for the next form of stomatitis, because it seems to represent most nearly the lesion which is seen on the mucous membrane, although it is not definitively settled that it is a true herpes.

The disease consists of a catarrhal stomatitis in the course of which certain lesions resembling subepithelial vesicles surrounded by areolæ occur irregularly and in different parts of the entire buccal cavity. This form of stomatitis has usually been known as stomatitis aphthosa (ἀφθα, an eruption or ulceration.) This name was given to it by Bohn as distinctive from the other forms of stomatitis, but it does not represent the affection especially well.

**ETIOLOGY.**—As a rule, when the mucous membrane of the infant's mouth is in a normal condition it is not readily affected by the various irritants which produce its special diseases. When a catarrhal condition is present the mucous membrane becomes more vulnerable and the various diseases have an opportunity to develop. This apparently is illustrated in the case of stomatitis herpetica, in conjunction with which affection a catarrhal stomatitis is always found. No cause, either local or general, has as yet been determined for this disease. Various micro-organisms have been observed in the mouth when it is affected by stomatitis herpetica, but no causal connection has been discovered between them and the disease. This affection may be found associated with a number of other diseases, but usually occurs alone. It does not seem to be contagious, nor to be especially connected with diseases of the gastro-enteric tract or with dentition, although it very commonly occurs during the dental period. It appears to be the result of certain deleterious influences which act upon the nerve-centres and produce an herpetic efflorescence on the mucous membrane which corresponds closely to that which is seen in herpes on the skin.

**PATHOLOGY AND SYMPTOMATOLOGY.**—In addition to the usual lesions of a stomatitis catarrhalis, spots, not necessarily symmetrical or unilateral, of different sizes and of different shades of white or grayish-white, appear in various parts of the mouth, especially on the inner surface of the lip, on the side and under surface of the tongue, and on the gums. These lesions do not affect the follicles of the mouth, and the efflorescence cannot be called follicular, as it is closely connected with the muciparous glands. The lesions make their appearance with great rapidity, and develop very quickly from a macule into what is supposed to be a vesicle. The action of the secretions of the mouth upon these lesions necessarily prevents them from having the same definite appearance that they would present on the skin. The course of the disease so strongly simulates that of herpes that at present it would seem wise to consider the efflorescence herpetic.

The general appearance of the efflorescence when at its height is that of a subepithelial vesicle, somewhat glistening, of a whitish-gray color, and surrounded by a red areola. The lesions may be only a few in number, scattered irregularly over the parts of the mucous membrane which I have already described. At times, however, the efflorescence is very diffuse, sometimes appearing as minute grayish points, which may become much larger and cover the mucous membrane so thickly as almost to simulate a false membrane. In a still later stage of the disease these lesions may break down and form small superficial ulcers.

An infant or young child affected by stomatitis herpetica presents a very characteristic appearance. It looks dull and apathetic, and wishes to lie quietly in bed. It usually has a heightened temperature, and evidently suffers from pain and heat in its mouth. The saliva flows from the mouth in large quantities, and often irritates the chin and neck to such an extent that an eczematous condition results. The child refuses to take its nourishment, and is very fretful and restless. These symptoms continue for four or five days or a week, and sometimes extend over a period of two weeks, the disease then disappearing of itself: in fact, it appears to be self-limited. Unless the lesions of stomatitis herpetica are complicated by those of stomatitis ulcerosa, the saliva is never fetid.

**PROGNOSIS.**—The prognosis of stomatitis herpetica is very favorable, although infection from other diseases may take place. This latter occurrence is, however, exceedingly rare. Relapses are very uncommon in this form of stomatitis, and the lesions usually heal readily.

**TREATMENT.**—There is no internal treatment which is of benefit in this disease. The indications for treatment are to allay the irritation of the mucous membrane and to prevent its infection by some other poison. The mouth in general should be treated as I have just recommended in the case of stomatitis catarrhalis. As a rule, very little treatment is necessary beyond occasionally cleansing the mouth with the solution (Prescription 74, page 779) already mentioned. The ulcers which do not heal readily can be touched with nitrate of silver. After the first few days, and earlier if



PLATE VII



*Trochus*



*Varicella*



*Franseria*



*Stomatia*



*Palmyra*



*Diphtheria*





the disease has attacked a puny, ill-nourished infant, great care and perseverance should be exercised to feed at regular intervals.

This boy (Case 388, Plate VIII., facing page 780, Stomatitis Herpetica), four years old, whom you see here in a darkened corner of the ward, is a pronounced case of stomatitis herpetica. He was perfectly well until two days ago, when he began to be feverish, was restless at night, refused to take his food, and seemed quite sick. On the following day the entire mucous membrane of the mouth was found to be affected with stomatitis catarrhalis, and somewhat later the herpetic form of stomatitis, which you now see in different parts of the mouth, appeared.

On drawing down the lower lip you see on the right side a number of small grayish-white spots surrounded by a somewhat deeper, reddened mucous membrane. At a little distance from them, on the left side of the lip, close to the gum, is apparently a subepithelial vesicle. On the inner side of the lower gum one of these vesicles has broken down, and a small superficial ulcer covered with a grayish-white exudation is seen. There are also lesions of the same vesicular character along the left edge of the tongue. The entire mucous membrane of the mouth is intensely reddened, and the case illustrates stomatitis catarrhalis as well as stomatitis herpetica.

The child absolutely refuses to take food, and, as he is robust, I have not advised that a great deal should be forced upon him. In a few days the more severe stage of the disease will have passed away and he will take his food. In the mean time the inflamed mucous membrane can be bathed with cold sterilized water, and small quantities of an alkaline modified milk can be given to him. As you look at this child lying with his eyes half closed, with flushed cheeks, in an apathetic condition, occasionally whimpering as if in pain, and with the saliva flowing continuously from his mouth on the pillow, you can readily diagnose the disease stomatitis. When in addition you see these characteristic lesions of the mucous membrane irregularly distributed throughout the buccal cavity, and do not find any evidence of a membranous exudation, there need be no doubt of the diagnosis. Internal remedies are not needed in a case of this kind. Chlorate of potassium, which is so commonly used in all diseases of the mouth, is not indicated in the forms of stomatitis of which I have just spoken.

In connection with this form of stomatitis may be mentioned certain lesions occurring in the mouths of new-born infants which have been called *Bednar's aphthæ*. These lesions consist of small superficial ulcers usually having a grayish coating, and appearing on the posterior part of the hard palate and on the soft palate. They are now supposed not to represent a specific disease, but to be the result of traumatism, such as may arise from a badly-shaped rubber nipple or from undue violence in washing the mouth.

They are to be treated as any local irritations of the mouth should be, —namely, by removing the cause, applying a solution of bicarbonate of sodium, and, if necessary, touching them with nitrate of silver.

**STOMATITIS ULCEROSA.**—By stomatitis ulcerosa we mean a peculiar pathological process of the mucous membrane of the mouth occurring only where there are teeth and affecting the gums around the teeth.

**ETIOLOGY.**—This affection of the mouth may occur in the course of a number of diseases, notably in scorbutus. It may also be produced by the internal administration of such mineral poisons as arsenic, lead, or mercury. Occasionally it may occur as a local affection without known cause, but it is probably produced by the irritation of some form of micro-organism not yet determined, although the pyogenic bacteria are very commonly present.

The most common form of stomatitis ulcerosa produced by the mineral poisons is that which is seen in connection with mercurial salivation.

As in the other forms of stomatitis, it is probable that the mucous membrane is first affected by a catarrhal process which renders it vulnerable to the special irritation which produces stomatitis ulcerosa. This preceding stomatitis catarrhalis may be produced directly by local irritation in the mouth itself, or may be the result of some disturbance of the general system. For this reason stomatitis ulcerosa, as a rule, does not affect primarily a healthy individual. Thus, a poorly nourished child, and one whose mouth is not properly cared for, will be more apt to have this disease develop than one who is correctly fed and whose mouth is clean.

**PATHOLOGY.**—The pathological condition is one of necrobiosis; that is, there is softening as well as death of the tissues. The disease, although starting in the mucous membrane, may extend to the periosteum, and even produce necrosis of the bone. It begins at the free border of the gums, and can extend in all directions, but it never passes beyond the mucous membrane of the mouth. The softening of the tissue not only changes its consistency but also renders it more movable, and in this way the gums at times become so swollen and loosened that they may entirely cover the teeth.

**SYMPTOMS.**—Stomatitis ulcerosa is usually preceded by moderate constitutional symptoms, such as fever, loss of appetite, and fretfulness. The mucous membrane of the gums at the free margin of the teeth becomes reddened and soon begins to swell. The normal curve of the gum becomes almost a straight line and covers the lower part of the teeth. The gums in the spaces between the teeth remain unaltered at first. The mucous membrane then begins to change in color and becomes purplish. Extreme congestion and softening of the tissues allow hemorrhage to take place from the slightest pressure. Although the anterior surface of the gums is most commonly affected, yet in severe cases the posterior surface is also involved. As the process develops further the gum becomes more and more loosened as it extends over the teeth. A muco-purulent secretion collects between the gum and the teeth and causes a fetid odor. According to Forchheimer, a yellowish seam then appears at the top of the swollen outline of the gum. This is due to the molecular destruction which has already begun. This seam is at first very narrow, but later it may become broader and involve almost the whole of the gum. In connection with this characteristic appearance of the gums there is a great hypersecretion of saliva. At the height of the disease the child evidently suffers from pain in the mouth, cries a great deal, and rapidly emaciates. The lymphatic glands are usually swollen, and remain so until the disease has ended. When the yellowish material which constitutes the seam already referred to is removed, an ulcerated surface will be found beneath. Although stomatitis ulcerosa may begin about any of the teeth, its most common starting-point is around the lower incisors. As the disease improves, the gums gradually become less swollen



and eongested, returning to their normal relation to the roots of the teeth, and the salivation disappears.

DIAGNOSIS.—The differential diagnosis of stomatitis ulcerosa when the lesions of the disease are marked presents no difficulty. Although an herpetic efflorescence may occur coincidently with the ulcerative form, yet the pictures of the two diseases are so different that you will at once know that you are dealing with two affections rather than with one. There is no other disease of the mouth in which the gums assume the purplish hue and the swollen, soft, and loosened condition which are characteristic of stomatitis ulcerosa.

PROGNOSIS.—The prognosis of stomatitis ulcerosa depends upon its cause and whether it is treated or not. The tendency is, however, after a variable period of diseomfort to the child, for the disease to disappear.

If the affection is the result of one of the constitutional diseases, such as syphilis or seorbutus, it disappears if the treatment of the specific disease is beneficial, otherwise it continues, and may finally lead to death by exhaustion.

TREATMENT.—The local form of the disease is best treated by the internal administration of chlorate of potassium or by this drug in solution used as a wash for the mouth. Chlorate of potassium must, however, be given with great precaution to infants and children, as in certain cases it acts as a poison, some infants being affected by even minute doses. The symptoms which show that chlorate of potassium is producing deleterious effects in infants who are most likely to be affected by the drug are drowsi-ness and suppression of urine, with weakness of the heart and sometimes cyanosis. When these symptoms follow the administration of the drug it should be omitted at once and a simple wash of borate of sodium used. Chlorate of potassium when given internally has been found to be secreted in the saliva within five or ten minutes, and thus has an opportunity of produeing a direct effect upon the lesions of the gums. The doses of chlo-rate of potassium which it has been found can be safely administered to infants and children should be remembered when prescribing the drug. I have indicated in this table (Table 108) the minimum doses which can safely be given in the twenty-four hours at different ages, and which are sufficient to produce the specific effect of the drug in treating cases of stomatitis uleerosa.

TABLE 108.

*Amount of Chlorate of Potassium which can be safely given in Twenty-Four Hours at Different Ages.*

Age.	Gramme.
Under 1 year . . . . .	1   0
1 to 2 years . . . . .	1   5
2 to 6 years . . . . .	2   0
6 to 8 years . . . . .	2   5
8 to 14 years . . . . .	3   0

In order that the chlorate of potassium shall produce the best effects it should be given frequently. The total amount for twenty-four hours which

is to be given at any special age is to be placed in a tumbler and dissolved in as many tablespoonfuls of sterilized water as there are doses to be given within the twenty-four hours. I usually tell the nurse to calculate about how many hours the child will sleep out of the twenty-four. Supposing the number of hours is ten: I then tell her to prepare fourteen tablespoonfuls of the solution and to give the child one tablespoonful every hour that it is awake. The administration of chlorate of potassium at first usually produces considerable smarting and pain in the mouth as it passes over the inflamed surface of the mucous membrane. These symptoms, however, last for only a short time, usually disappearing entirely after from thirty-six to forty-eight hours.

Under this treatment the disease is ordinarily cured in a week or ten days. The treatment should, however, be continued for a number of days after the mouth is apparently entirely well.

Where deeper ulceration has taken place, its disappearance may sometimes be expedited by the application of nitrate of silver. Where a sequestrum has formed, it must be removed. Frequent washing of the mouth with sterilized water administered by means of a dropper is also very important, especially after the taking of food. An alkaline diet is indicated.

I have here an infant (Case 389, Plate VIII., facing page 780, Stomatitis Uleerosa in Scorbutus), ten months old, in whose mouth you will see the characteristic lesions of stomatitis uleerosa. In this case the disease happens to be secondary to scorbutus, the affection for which the infant is being treated.

You will notice that the infant has six teeth, and that the mucous membrane is affected only at the junction of the gums with the free surface of the teeth. The other parts of the mucous membrane of the mouth are reddened, but not markedly so. The portions of the gums affected are swollen, purplish, loosened, and almost cover the teeth. There is a considerable flow of saliva, with a fetid odor from the mouth. An appearance of this kind is diagnostic of stomatitis ulcerosa.

I also have here a case of stomatitis ulcerosa which apparently is of local origin. This little girl (Case 390) is three and a half years old. She has always been healthy, and has had no diseases of any kind. She was perfectly well until five days ago, when she began to have loss of appetite, a temperature varying from 38.3° to 39.4° C. (101° to 103° F.), and to be very fretful. Three days later the gums were noticed to be swollen and to be of a dark red color, and her breath had a fetid odor. During the past two days 1.5 grammes (25 grains) of chlorate of potassium have been given to her in divided doses in the twenty-four hours, and, although she has been rather apathetic and has wished to remain in bed, her mouth to-day is in a much healthier condition, and she is brighter and has a little return of appetite.

In two or three days more the disease will probably have run its course and entire recovery will have taken place. The salivation, which was very marked in the early days of the disease, is now quite moderate.

During the first three days her restlessness was so excessive at night that 0.3 gramme (5 grains) of bromide of potassium had to be given to her to produce sleep.

**STOMATITIS MYCETOGENETICA.**—There are three forms of vegetable parasites which occur in or upon the human body: (1) bacteria, or fission-fungi (schizomycetes); (2) yeasts, or yeast-fungi (saccharomycetes); (3) moulds, or mould-fungi (hyphomycetes). The changes in the tissues



which are due to fungi are termed mycetogenetic metamorphosis, and thus the pathological conditions in the mouth which are produced by any of these forms of fungi may be designated by the general term mycetogenetica. Under this general heading of mycetogenetica we can include the various forms of stomatitis which are caused by fungi.

STOMATITIS HYPHOMYCETICA (Thrush).—The disease which is commonly called thrush is produced by a fungus which finds its nidus upon the surface of the mucous membrane of the mouth, usually in young infants. This fungus was formerly supposed to be the *oïdium albicans*, but it is now known not to be this organism, and the precise form of mould which it represents has not yet been determined. We merely know that this growth of thrush is one of the mould-fungi, and we can therefore at present only classify it as *stomatitis hyphomycetica*.

The moulds are complex in their structure, and as commonly described consist of a series of delicate jointed threads (mycelium) in which spores are developed. Hyphomycetic growth is characterized by having the spores naked on conspicuous threads. The fungus of thrush may be found on any of the mucous membranes of the body. It has also been found in various organs, as in the brain and the lungs, and from the surface of ulcers it has on rare occasions penetrated the blood-vessels and given rise to visceral metastasis. The usual place for it to appear, however, is the mucous membrane of the mouth. It is a local disease, and may occur in the mouths of healthy children as well as in those who are diseased. It is more likely, however, to be ingrafted upon a diseased than upon a healthy mucous membrane, in accordance with the rule which I have already stated. A catarrhal condition of the mucous membrane, by displacing the epithelial cells and thus interfering with their protection of the mucous membrane, affords the readiest means for the development of the fungus of thrush. It is therefore more likely to be found in the mouths of children who are suffering from various diseases or who are ill cared for. It may be carried to the mouth in various ways, either on dirty nipples or by the finger.

PATHOLOGY.—The growth may take place on both squamous and cylindrical epithelium. According to Forchheimer, the first lodgement of the fungus comes between the epithelial cells of the mouth, and from this the growth works its way under the free surface of the mucous membrane. When directly on the free surface the growth is not so luxuriant and is principally in the mycelium form. In the case of a mucous membrane lined by flat or squamous epithelium, the growth is facilitated by the relation of the cells to one another. In a membrane lined by cylindrical epithelium the growth takes place, but not so readily, because there is but one layer of cells. After the first development the growth goes on very rapidly, and after it has found a nidus the cells are pushed aside and are surrounded by mycelium, the whole presenting the characteristic appearance of thrush. The growth begins in small spots, sometimes one, sometimes more, and at times the entire surface of the mucous membrane is covered with it. The

fungus develops within the epithelium, and it requires considerable rubbing to remove the growth from the mucous membrane.

**SYMPTOMS.**—An attack of thrush usually begins with local symptoms of catarrhal stomatitis. At times, however, no symptoms are present, the fungus being the first abnormal condition which is noticed. The appearance of the fungus resembles closely that of curdled milk, though it is often of a rather grayish color. It does not look like a membranous exudation, but is raised in small patches above the level of the mucous membrane. The fungus usually develops on the inner borders of the lips, on the gums, on the tongue, and on the hard and the soft palate. It may extend to the tonsils and pharynx, and even into the œsophagus. In the latter locality at times it has been found to grow so thickly that the lumen is almost entirely occluded. The local symptoms are commonly those of a mild catarrhal stomatitis. The general symptoms depend upon the extent of the local disease from which the infant is suffering. Infants affected with this disease soon become atrophic, from a lack of proper nourishment, as they are often unwilling to take their food or cannot swallow it without difficulty.

**DIAGNOSIS.**—The differential diagnosis is seldom difficult to make. Curdled masses of milk on the inner surfaces of the lips and on the gums may resemble closely the fungus of thrush, but the former is easily wiped away, while the latter is difficult to dislodge. The disease is definitively determined by placing some of the growth under the microscope, where it presents characteristic appearances which I shall presently show you.

**PROGNOSIS.**—The prognosis of thrush varies according to the general condition, the vitality, and the age of the subject on whom it is engrafted. The disease may last indefinitely if the mouth is not carefully treated, and its prolongation may render the prognosis more grave. Where the growth is very extensive, as in the cases where it has invaded the œsophagus, the prognosis is very unfavorable. In these cases disturbances of the gastro-enteric tract are apt to arise and to increase the likelihood of a fatal issue. As a rule, however, if the infant's health can be maintained, and if the local treatment is carried out thoroughly, the prognosis is favorable.

**TREATMENT.**—The treatment should be directed to the local care of the mouth and to supporting the strength by proper nourishment and stimulants until the fungus has been eradicated. Care should be taken that everything connected with the infant, especially the nipples and bottles from which it is to be fed, should be aseptic, so that it shall not be continually reinfected or infect other children. The mouth after each feeding, and also between the feedings, should be thoroughly and somewhat vigorously rubbed with the solution (Prescription 74, page 779) which I have already recommended in the treatment of stomatitis catarrhalis.

Where the disease is in the œsophagus it is best treated by the introduction of a soft rubber tube, in order that the growth may thus be mechanically separated from the mucous membrane.

In many cases the disease is very intractable. No special drug appears



to be of use in these cases, and they can be cured only by the unremitting and patient removal of the growth as I have just described.

I have here an infant (Case 391, Plate VIII., facing page 780, Thrush), three months old, who has refused to take the bottle for the past month, is emaciated and fretful, and at times vomits.

A careful physical examination fails to detect anything abnormal except in the infant's mouth. On gently depressing the tongue and lower jaw, it is seen that the soft and the hard palate, the tongue, the gums, and the inner surface of the lips are covered almost entirely with white and grayish-white masses, in texture somewhat resembling curdled milk, and rising above the level of the epithelium. Between these patches the mucous membrane is reddened. There is a moderate flow of saliva. This morbid growth apparently does not extend into the pharynx. On endeavoring to remove one of these patches you see that it cannot be done readily, as would be the case if it were curdled milk, but that it has evidently passed between the epithelial cells down to the underlying mucous membrane, where it is held so closely that it requires considerable rubbing to separate it. In this case the growth is so extensive that it simulates a membrane in some places, but its generally roughened surface, its elevation above the level of the mucous membrane, and the characteristic appearances in other parts of the mouth render its recognition quite easy.

On placing some particles of this growth in glycerin under the microscope (Fig. 98), you see a tangled mass of fine, almost translucent, membered threads.

CASE 391. FIG. 98.



Mycelium of thrush interspersed with spores and fatty degenerated cells. (Low power Zeiss Oc. 3, Objective DD, glycerin.)

Interspersed among these are bright, glistening, oval bodies, which are the formed spores, and also fatty degenerated cells and fine detritus. This combination of appearances represents the pathological processes which we find in thrush.

Under this second microscope (Fig. 99, page 788) you will see some shreds from the same specimen, but much more highly magnified.

In this specimen you can see the formation of the spores in the mycelium.

Under this same heading of stomatitis mycetogenetica I shall merely refer to those pseudo-membranous conditions which occur in diphtheria,

tuberculosis, syphilis, and diseases of a like class. The former two are so rarely seen in the mucous membrane of the mouth that it is not necessary to describe them. The lesions which occur in the mouth in syphilis I have already described when speaking of that disease (page 494).

CASE 391. FIG. 99.



Thrush showing the formation of spores in the mycelium. (Zeiss Oc. 3, homogen. immer. 2.0 mm.)

**STOMATITIS GANGRÆNOSA (Noma, Cancrum Oris).**—Stomatitis gangrænosa is the rarest and most fatal form of stomatitis which occurs in children. It is usually met with between the ages of three and seven years. It is a disease characterized by a gangrenous process which begins on the gums or on the inner surface of the cheek and spreads with great rapidity to the adjoining tissues, all of which can be involved and quickly destroyed.

**ETIOLOGY.**—It is probable that there is a specific germ which causes this disease. This organism has, however, not yet been determined. It is supposed that it does not attack a healthy mucous membrane, and that one of the other forms of stomatitis, especially stomatitis catarrhalis, and in some cases stomatitis ulcerosa, precedes it. Furthermore, stomatitis gangrænosa seldom attacks healthy children, but usually affects those who have other diseases and are greatly debilitated. It occurs most commonly secondarily to the acute exanthemata, especially measles. The disease is also said to result from the administration of mercury in too large doses.

It begins as a reddened, hard spot in the mucous membrane, usually of the cheek. This soon becomes gangrenous and extends rapidly through the entire thickness of the cheek, producing perforation. It may also



extend laterally in all directions, attacking the bone as well as the other tissues.

**SYMPTOMS.**—The first symptom which is apt to be noticed is the gangrenous odor which comes from the mouth. On examination an ulcer will be found which tends to spread rapidly. The cheek becomes much swollen, is hard and œdematous, the œdema especially affecting the tissues under the eye. The gangrenous process extends very rapidly, at times destroying large portions of the face, and also involving the bones, which become denuded. The teeth become loose and fall out. The odor from the gangrenous tissue is excessive. The flow of saliva is very much increased. The degree of suffering which the children undergo varies very much: sometimes it seems as if they suffered no pain whatever. The temperature varies, at times being raised and again being subnormal. The pulse is weak and rapid. The appetite is diminished, and the children are likely to have diarrhœa, probably due to the infectious nature of the products of the mouth which are swallowed. Hemorrhages are rather rare, according to Forchheimer, as the blood-vessels are usually filled with thrombi. Secondary affections, such as catarrhal pneumonia from the inhalation of septic material, are not uncommon. The child may die from one of these secondary affections, or it may become more and more weakened by the local condition, and unless the morbid process is arrested it will die eventually from exhaustion.

**DIAGNOSIS.**—The diagnosis of this disease, except in its earlier stages, is not difficult. At times, however, a local ulcerative process produced by a decayed tooth may simulate closely stomatitis gangrænosa. In these cases the diagnosis is made more difficult by the fact that the tissues of the cheek may become hard and look as though perforation might take place. Coincidentally with this condition the ulceration of the gum and often of the mucous membrane of the cheek, with the foul odor which emanates from it, makes the similarity of the two diseases very striking. In simple ulceration from a tooth, however, active local treatment with solutions of myrrh or of soda combined with frequent washing of the mouth with sterilized water is soon followed by marked improvement, while where stomatitis gangrænosa is present the morbid process continues to extend.

**PROGNOSIS.**—The prognosis in cases of stomatitis gangrænosa where they are untreated is almost universally fatal. Cases have been known, however, where a line of demarcation has formed around the gangrenous spot, granulations have arisen, and cicatrization has followed, leaving extensive scars. If the disease is treated by extirpation of the diseased structure in the very beginning, the prognosis becomes more favorable. Where the disease has perforated the cheek and the gangrenous process has become extensive, the child is seldom relieved even by surgical treatment.

**TREATMENT.**—Care should be taken when a child is affected with a disease of an exhausting nature that its mouth is kept thoroughly cleansed, for we can never tell when or in what individual the mucous membrane

may become vulnerable to the organism which produces stomatitis gangrenosa. In stomatitis gangrenosa it is very important for the success of the treatment that it should be begun very early in the disease. Where the diagnosis has been definitively made, it is wiser not to temporize with applications of nitrate of silver and other drugs, but at once to place the case in the hands of a surgeon and have the entire area of the invaded tissues excised. It is also well after the gangrenous process has been removed by the knife to destroy an area of healthy tissue by means of the Paquelin thermo-cautery or by the galvano-cautery. There should be no delay in operating upon these cases, as great destruction of the tissues may take place in even a few hours.

After the operation the tissues should be inspected frequently, to see whether there is any return of the gangrenous spots, and, if found, these spots should be removed immediately. As the disease is very apt to return, plastic operations to obviate deformity should not be undertaken very early after the operation.

In treating these cases surgically it must be remembered that the child is in a very debilitated condition, and that if it is suffering from any especial disease treatment directed to that disease is indicated, also that stimulants are required to prevent the already weakened child from dying of exhaustion following the operation.

Here is a little girl (Case 392), four years old, who has been brought to the hospital to be operated on for stomatitis gangrenosa.

#### CASE 392.

##### I.



Stomatitis gangrenosa, left cheek (before operation). Female, 4 years old.

In this case the disease was apparently primary, and began on the the left side of the mucous membrane of the mouth. It spread rapidly, and, although treated by local applications to the mouth with various solutions, has now, as you see, broken through the left cheek close to the ala nasi. The teeth are loose in the middle of the upper jaw, and there



is a certain amount of alveolar necrosis. There is a strong gangrenous odor from the mouth and the tissues of the cheek, and a considerable flow of saliva. The child's general condition is fair, but she is becoming more debilitated, has lost her appetite, and has a slightly raised temperature. The operation should be performed immediately.

## CASE 392.

## II.



Stomatitis gangrenosa, left cheek (after operation).

(Subsequent history.) The cheek was operated on the day after the child entered the hospital, by Dr. H. W. Cushing. The wound healed readily, and this picture (II.), taken some months afterwards, shows the scar on the cheek close to the ala nasi and also on the upper lip.

## CASE 392.

## III.



Stomatitis gangrenosa, right cheek (before operation).

One year later the child again returned to the hospital, and on examination was found to present the appearances which are seen in this picture (III.), taken at that time.

The right cheek was much swollen and indurated, especially under the right eye. The periosteum of the lower jaw on the right side was found to be affected, and the necrotic process had undermined the whole cheek as far as the orbit. The child was operated on by Dr. Bradford without any external opening of the cheek. The wound healed, and the child was discharged from the hospital, but returned some months later with a spontaneous opening on the right cheek. This was again apparently cured by operation. Two months later the child was found to have in the lower jaw a process similar to that which had occurred in the upper jaw. Her health was poor, she was pale and weak and had loss of appetite. She was operated upon again, and a sequestrum was removed from the lower jaw. She then improved, and this picture (IV.) was taken some months later, when she was apparently in fair health.

## CASE 392.

## IV.



Stomatitis gangrænosa, right cheek (after operation).

The microscopic examination of the gangrenous tissues removed at the operation presented nothing significant of any especial disease, and a culture made by Dr. Stone showed only a few streptococci.

You will remember the case of measles (Case 257, page 587) complicated by stomatitis gangrænosa which I showed you at a previous lecture, and the result of which I now report to you.

As I told you at that time, the disease was preceded by pertussis, measles, and a broncho-pneumonia. After she had the pneumonia for seventeen days her right cheek began to swell and a bad odor to come from her mouth, but nothing especial could be found in the mucous membrane of the buccal cavity. Four days later the swelling of the cheek had much increased, and there was œdema of the lips and eyelid so that the right eye was partly closed. The swelling was semi-fluctuating. The temperature varied from 38.3° to 39.4° C. (101° to 103° F.), and the cough had much lessened. On the following day a bluish-black spot about 1.5 em. ( $\frac{3}{4}$  inch) in circumference appeared at the right corner of the mouth, and this rapidly increased during the day. Two days later the dark-colored area had increased considerably in size and presented a circular outline with a clearly marked line of demarcation.

The child also had a profuse greenish diarrhœa. On the following day the dark area rapidly extended, and soon involved the whole of the right cheek, the right side of the mouth, and the right nostril. There was no external loss of tissue. The child was extremely emaciated, and from the beginning of the attack was in a hopeless condition, so



that radical treatment of the disease was deemed inadvisable. It died suddenly on the following day.

## CASE 257.



Stomatitis gangrenosa secondary to measles and pneumonia. Female, 5 years old.

**GLOSSITIS.**—Glossitis is so rare a disease in children that the possibility of its occurrence only need be mentioned. In this affection there is an acute inflammation of the tissues of the tongue, accompanied by fever, enlargement of the organ, and considerable pain. There is usually a hypersecretion of saliva, and at times the obstruction of respiration from the occlusion of the throat by the greatly enlarged tongue produces somewhat alarming symptoms, though, as a rule, not serious ones.

This disease may be caused by direct injury to the tongue from corrosive substances, by heat, or by the stings of animals, and sometimes probably by sepsis. It runs a variable course; it is not especially serious, and tends to recover after a few days. The treatment is purely symptomatic. The frequent local application of ice and of ice-cold alkaline solutions to the tongue and mouth is indicated.

A condition of the dorsum of the tongue is sometimes met with which for want of a better name is called *lingua geographica*, “mappy tongue,” or “wandering rash.” One or more small patches appear on the dorsum or side of the tongue, which in a few days may spread and coalesce, covering often a large portion of the surface. They diminish in size or fade with equal rapidity, to recur at variable periods. The patches are red and smooth, and the filiform papillæ are absent. The rest of the tongue appears normal, except that the papillæ on the borders of the denuded portions are white and prominent. The etiology of the disease is unknown. It occurs almost exclusively in children or in young adults who have been subject to it from childhood. It is very benign, and gives no discomfort to the child. Its principal importance lies in the fact that it is sometimes mistaken for a symptom of some more serious disease. No form of treatment has been

found useful. It recurs periodically for months or years, but does not tend to increase in severity nor to lead to other diseases.

**MICROGLOSSIA.**—In some individuals an arrest of development of the tongue produces the condition called microglossia, in which the tongue is to a varying degree smaller than normal.

**MACROGLOSSIA.**—The opposite condition, macroglossia, in which the tongue is enlarged, is more common than microglossia. It is usually a congenital lesion, and is especially marked in cretins. The prominent feature of the affection is a prolapse of the tongue, which is often enormously enlarged in every direction, is usually of a deep violet color, and is covered with a thick, whitish coat. The protruded tongue is indented and even ulcerated by the teeth, which are often pushed forward and become carious. The saliva flows continuously from the mouth, the lower lip becomes thick and ulcerated, and the forcing forward of the lip, larynx, and velum palati by the weight of the tongue renders suction, mastication, and deglutition difficult. The nutrition of the child is thus much interfered with, and this interference is one of the most serious results of the disease. This condition is not a glossitis, but a deformity which seems to be associated with certain other malformations of the body. In these individuals the hands and feet are apt to be large, thick, and purplish.

Macroglossia appears in two forms. One is the fibrinous, in which the connective tissue is pathologically increased between the muscular fibres. The other is a cavernous cystoid degeneration of the interstitial connective tissue, by which the resulting spaces come into connection with the lymph-vessels, constituting a condition closely resembling cavernous angioma, from which it receives its name of lymphangioma cavernosum.

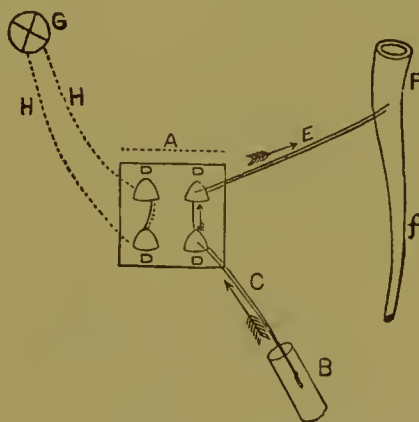
The disease seldom tends to recover, and the treatment is to give as much relief as possible to the great discomfort which arises from it, by cleansing the mouth frequently with alkaline solutions. Especial care should be directed to the nourishment of the child. In extreme cases surgical interference is indicated where the child's respiration and general nutrition are affected, and in some cases great improvement is accomplished by the removal of part of the tongue.

**DIFFICULT DENTITION.**—I have already described to you the process of the normal development of the teeth in infancy and childhood, and have impressed upon you that this process is a physiological one. The teeth are developed at birth to a certain degree, and merely increase in size during infancy until they pierce the gums and assume their places in the mouth. In many cases the process of dentition gives rise to no morbid conditions whatever. The idea that dentition occasions the various diseases with which it was formerly supposed to be associated is an erroneous one. From the fourth or fifth month, however, until the completion of dentition in the latter part of infancy, various nervous disturbances are so closely associated with irritation in the mouth that in this sense dentition may be considered responsible for many of the slight ail-



ments which arise at this period of life. The mouth at this time frequently becomes hot, and sometimes dry, although there may be a hypersecretion of saliva. There is evidently much discomfort in the region of the gums, as the infant is continually rubbing them with its fingers and seems to get relief from biting on hard substances. Such infants may become much prostrated and may lose their appetite, and thus their nutrition may be interfered with, without any discoverable cause for these abnormal conditions beyond the general nervous irritation which arises from the feeling of discomfort in the mouth and head. In the more extreme cases the infant will be so restless at night that it scarcely lies still for half an hour at a time, and may spend night after night crying out occasionally as though in pain, and knocking its head against the sides of its crib, so that in some cases the crib will have to be padded. These infants also have to be guarded sometimes from knocking their heads against the floor or wall, as they seem to become almost frantic from the continued irritation from which they are suffering. These symptoms occur with such regularity at a time when a tooth is in its final stage of development, and cease so uniformly when the tooth has attained its growth, that the causal relation between the tooth and these nervous symptoms seems more than probable. This rather indefinite clinical association of dentition and nervous symptoms is, however, partially explained by the analogous symptoms arising from the anatomical relationship which exists between the roots of the teeth and the ear. It has long been noticed that in certain individuals during the completion

DIAGRAM 10.



A, sympathetic ganglion; B, sensori-motor nerve; C, afferent sympathetic fibres from sheath of B; D, caudate cells; E, efferent sympathetic fibres proceeding to artery *f*; F, artery dilated; *f*, normal size of artery beyond the sympathetic influence; G, general vaso-motor centre; H, H, the dotted lines indicating the course of the fibres forming the roots of the ganglion in the spinal cord to the general vaso-motor centre G. (Woakes.)

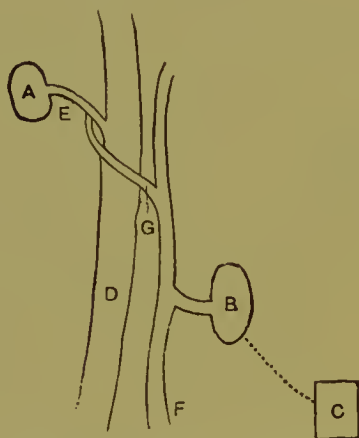
of the development of a tooth symptoms connected with the ear will manifest themselves. These symptoms are usually produced by congestion of the blood-vessels of the ear, which is accompanied by pain, and sometimes results in inflammation. They are evidently of reflex origin. If you will study this diagram (Diagram 10) you will understand the influences which an irritation of some distant part of the economy may exert on the blood-vessels of the ear.

The general vascular disturbance in the ear, represented either by an uncomfortable feeling of fulness or by general pain, may be produced in cases of difficult dentition by this close connection between the sensori-motor nerves and the sympathetic. According to Woakes, a considerable portion of the blood-supply of the membrane of the drum is derived from the artery that leaves the internal carotid in the carotid canal and proceeds by a very short course directly to its destination. Being thus closely connected with a large arterial trunk, this small tympanic branch is very favorably situated for a speedy augmentation of its blood-supply. The nervi vasorum constituting the carotid plexus at this part of its course come largely from the otic ganglion. On the other hand, the inferior dental nerve supplying the gums and the teeth also communicates with this ganglion.

We thus arrive at a direct channel of nerve communication between the source of irritation in the mouth and the vascular supply of the drum-head. The earache which arises in these cases is produced by the vessels of the membrana tympani, which become greatly distended, and the accompanying stretching of the tense and sensitive tissue in which this occurs accounts for the pain.

I have represented in this diagram (Diagram 11) the anatomical nervous connection between the teeth and the membrana tympani.

DIAGRAM 11.



A, tympanic cavity; B, otic ganglion; C, tooth; D, internal carotid; E, tympanic branch; F, auriculo-temporal nerve; G, auricular branch of auriculo-temporal nerve. The dotted line connecting B and C represents the inferior dental nerve.

You will thus see that a great many symptoms, usually of slight import, but marked enough to give much discomfort to the infant, may arise during this period of dentition, when the infant's entire nervous system seems to be in a very sensitive condition.

**GUM-LANCING.**—The question of lancing the gums during the period of dentition is one which has given rise to much discussion and to very diverse opinions. In former times it was erroneously believed that the teeth played an important part in almost every disease which occurred in early life. It was also supposed that lancing the gum relieved the symptoms of these diseases in some unexplained way. This extreme view soon had to be modified, and of late years many observers have come to the



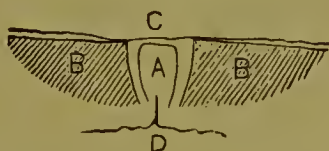
conclusion that it is never necessary to lance the gums. In cases of difficult dentition, however, as I have just explained, irritation arises very commonly in the later stages of the development of a tooth, and the question, therefore, remains whether this irritation in various parts of the economy, notably in the ear, can be relieved by lancing the gum. With regard to the question of gum-lancing, it may be said that it should be resorted to only under very exceptional circumstances.

During the dental period two classes of irritation are met with in connection with the teeth: (1) irritation of the dental nerves, with symptoms of reflex aural disturbance; and (2) irritation of the gum over the crown of the tooth from pressure, with symptoms of local irritation. We here have two entirely different conditions. If, where pain or symptoms in some other part of the economy seem to arise from dental irritation, we find that the gum which covers the crown of the still undeveloped tooth is soft and flat as in other parts of the mouth where a tooth is not about to come through, lancing the gums is manifestly absurd, as there is evidently no reason for making a wound in the mouth.

The second class of cases, however, though exceedingly rare, must still be recognized as distinct in themselves and requiring especial treatment. In this class it is very evident that the gum for some reason does not give way to the growth of the tooth. Where the gum covers the crown of the tooth the tissues are swollen, tense, almost cartilaginous in their feeling, and hot. As in like conditions, either in the mouth or elsewhere, when this combination of abnormal conditions is found over the crown of the tooth, it can be relieved at once by the lancet.

I have here two diagrams which represent the condition of the gums in relation to the teeth in the two classes of cases which I have just mentioned. In this first diagram (Diagram 12) you see that the mucous membrane over the crown of the tooth is flat and on a level with the rest of the gum.

DIAGRAM 12.



A, tooth in bone socket; B, jaw; C, gum, soft, not inflamed or swollen; D, dental nerve.

This is the condition of the gum in the majority of cases of difficult dentition, yet very severe symptoms of disturbance of the ear and cerebral circulation may apparently arise in these cases. The symptoms, of course, are very varied, the most definite ones being connected with the ear. In this class of cases the gum should never be lanced, even for the purpose of bleeding, as the mouth is not a fit place for such a procedure. The treatment of these cases should be directed to the especial part of the economy from which the symptoms arise. For instance, if the ear is affected, the indication is to relieve the reflex congestion. This can be done by the instillation into the ear of a few drops of an atropine solution (Prescription 75).

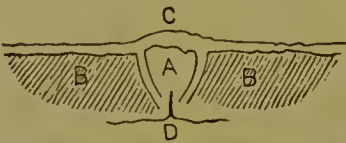
PRESCRIPTION 75.

<i>Metric.</i>		<i>Gramma.</i>		<i>Apothecary.</i>
R Atropinæ sulphat. . . . .		0   06	R Atropinæ sulphat. . . . .	gr. i;
Glycerini,			Glycerini,	
Aq. destil. . . . .	aa 3	75	Aq. destil. . . . .	aa ʒi.
M.			M.	
Sig.—Drops for aural congestion.				

In addition to this, bromide of potassium should be given in repeated doses to the extent that is indicated by the especial case.

In this next diagram (Diagram 13) you will see that the mucous membrane covering the crown of the tooth is markedly raised above the level of the gum.

DIAGRAM 13.



A, tooth in bone socket ; B, jaw-bone ; C, gum, tense, inflamed, swollen ; D, dental nerves.

In these cases, symptoms of local origin and often of great severity arise. The infant evidently has extreme pain and tenderness in its mouth. It cries incessantly, and often refuses to take its nourishment, on account of the acute pain which it suffers, and also of the tenderness which is produced

by the least pressure on the gum, so that it may become weak and exhausted. There is usually a considerable heightening of the temperature, to 38.8° C. and even 39.4° and 40° C. (102°, 103°, and 104° F.). Vomiting is not uncommon, and there is twitching to such an extent that convulsions seem to be threatening, and at times actually occur. There are also great restlessness and insomnia.

In these cases lancing the gum produces immediate relief. The temperature quickly goes down, the pain and general nervous symptoms disappear, and the infant after sleeping quietly for an hour or so wakes up very hungry and takes its food with avidity. The treatment in this class of cases, when the diagnosis is once made, is evidently to lance the gum. This is done in the following way. The infant is placed in the nurse's lap, with its head in the lap of the physician, the nurse holding its arms firmly. The physician, after having first thoroughly sterilized his hands and washed the infant's mouth and gums with sterilized water, carefully makes an incision over the swollen gum well down to the crown of the tooth. I have here a lancet (Fig. 100) which I am in the habit of using for this purpose.



Gum-lancet.

As only the end of this lancet is sharp, there is less danger of wounding the infant's lips and mouth than when using the ordinary bistoury. Before using the lancet it should be thoroughly sterilized.



Although much has been said about the danger of hemorrhage in these cases, and of infection of the wound by pathogenic organisms, yet instances where such results have occurred are so exceedingly rare that they should not deter us from treating the case properly as we would treat an abscess in the mouth, tonsil, or pharynx. It has also been said that a cicatrix may form on the gum over the crown of the tooth as a result of lancing. This is an exceedingly rare occurrence, and need scarcely be taken into account. The probability is, where such an instance has occurred, that the case was not one in which the gum should have been lanced, and the fear of such a result as this should certainly not weigh in the balance against the possible exhaustion and acute pain which may continue for days unless relief is given by cutting.

I have a number of cases to show you which will serve to illustrate what I have endeavored to impress upon you in speaking of *difficult dentition*,—namely, that the indications for lancing the gums very seldom arise.

This infant (Case 393), ten months old, has been brought to the clinic with the following history:

It has one lower incisor. At the time when this tooth was about to appear above the margin of the gum the infant was very restless, and had considerable fever, and pain in its ear. Somewhat later a muco-purulent discharge came from the ear, but the general symptoms of restlessness, pain at times, and the local symptoms of heat and irritation in the mouth continued until just before the tooth had pierced the gum. After that time, which was three weeks ago, the discharge from the ear ceased, and the infant became perfectly well, the local irritation also having disappeared.

During the last three or four days, however, the same symptoms have returned. The infant is evidently suffering from irritation in its mouth. Sometimes the gums are hot and dry, and again there is a hypersecretion of saliva. It continually puts its finger to the gum of the lower jaw, sometimes almost locating it near the place where the first tooth has been cut. The ear has begun to discharge again, and the infant shows signs of general discomfort by rubbing its nose and head continuously and at times crying out as though in pain.

On examining the gum you see that it is not swollen, and that there is no especially tender point. On examining the ears an old perforation of the membrana tympani is found in the right ear, which is discharging, while in the left ear there is a simple congestion.

Such cases as this are often treated by lancing the gum, yet this procedure is not of the slightest use,—is, in fact, contra-indicated, as it will only increase the already existing irritation of the mouth. The treatment is the internal administration of bromide of potassium and appropriate local treatment for the ear.

The other cases are so similar and are so commonly met with that I need not dwell upon them, but shall report one of the rare cases in which lancing of the gum is indicated.

An infant (Case 394), eight months old, and in good health, cut its first tooth when it was seven months old. At this time there were no nervous disturbances, the tooth coming through the gum without any reflex or local symptoms whatever.

When the second tooth was pressing on the gum I was called to relieve the following symptoms. The infant, who had been perfectly well, and who on examination showed no disease of any organ, was reported to have been feverish, restless, and crying out with pain for the previous twenty-four hours. It had refused to nurse, had not slept for thirty-six hours, had vomited a number of times, and was found to have a temperature of 40° C.

(104° F.). It twitched from time to time, and apparently was in danger of having general convulsions. On examining the mouth I found that one of the lower middle incisors was entirely through the gum. The gum next to this incisor was greatly swollen, tense, cartilaginous in feeling, hot, and tender, so that whenever it was touched the infant screamed with pain. I then lanced the gum. The expression of pain, which had been most marked on the infant's face, disappeared immediately, and was replaced by an expression of perfect tranquillity, and it was evident that the severe pain had been relieved instantaneously. The infant went to sleep at once, and slept two hours. When it awoke its temperature was normal, it took the breast with great eagerness, and from that time it had no more trouble in its mouth. All the rest of its teeth were cut without any abnormal symptoms.

I have also to report to you another instance which illustrates to a still greater extent the necessity of lancing the gums in certain cases.

An infant (Case 395) began to have irritation from its teeth when it was five months old. At this time it woke up in the night screaming, and continued to scream with pain for several hours, during which time its parents had to walk continually up and down the room with it. Various remedies were administered, but without the slightest relief, and finally, after two days of suffering, in which it refused to take its nourishment, it lost in weight, and seemed very ill. An incision was made over the hot and swollen gum, with immediate relief.

The same symptoms occurred when the next tooth appeared beneath the surface of the gum, but were relieved, after waiting for a few hours, by lancing. Of the remaining eighteen teeth, six or eight gave rise to similar symptoms, but in every instance immediate relief was afforded by the lancing of the gum.



## LECTURE XLI.

## DISEASES OF THE NOSE, NASO-PHARYNX, AND PHARYNX.

**NOSE.**—The nose is the normal passage for the entrance of air to the lungs, and it is principally here that the air is modified before entering them. In normal respiration the mucous membrane of the nasal cavities, on account of the peculiar shape of the turbinated bones, presents a large surface to the inspired air, and is therefore admirably adapted to filter it of particles of dust and micro-organisms. The air is also warmed and changed so that before it reaches the larynx it is saturated with moisture and heated to a temperature of 35° C. (95° F.). This modification of the air is especially important in the new-born, since the lung has so lately been brought into use and is in such a comparatively undeveloped condition that it cannot withstand unchanged air, to which it adapts itself better later in life. I have already described to you (page 33) the extremely narrow passage through which the air passes in going to and through the naso-pharynx in young infants, and how easily this passage can become occluded. There are not many diseases which occur in the nose in infants and young children, and those which we find are serious chiefly by being the cause of occlusion. In case of mouth-breathing due to nasal occlusion in an infant, the air which has not been modified by passing through the nose and naso-pharynx may have a detrimental influence on the lung and general circulation, thus striking a serious blow at the infant's vitality. In later childhood, although the occlusion which arises in the nares may not be so serious as regards the life of the patient, yet you will see the results of such a condition represented by retarded development of the child and interference with the function of hearing, with its resulting mental dulness.

The most common pathological condition which occurs in the nose in infancy and childhood is some form of *rhinitis*. This may be acute or chronic, catarrhal or purulent, hypertrophic or atrophic. New growths are rare. Of these the more common is myxoma or simple mucous polypus. Bleeding from the nose, called epistaxis, may arise from an ordinary non-inflammatory condition, and is generally due to the breaking of a superficial vessel on the septum.

**ACUTE RHINITIS (Acute Coryza).**—Acute rhinitis is an inflammation of the mucous membrane of the nasal cavities. The cause of the disease in most cases is apparently undue exposure to cold, though it may be proved eventually that this exposure merely prepares the way for the attack of some micro-organism. This condition may in almost all cases be considered as part of a disease which affects the mucous membrane of the naso-pharynx and pharynx as well as the nares.

The symptoms are a sense of fulness, burning, and dryness in the nostrils, succeeded in a few hours by a serous discharge, which later becomes muco-purulent. There is usually a slight rise of temperature, and, although the general symptoms are often slight, there is commonly a very evident sense of discomfort, along with loss of appetite and general malaise. In some cases, by direct extension of the inflammation through the Eustachian tubes, an otitis media may be caused. Especially in young infants, the entrance of air into the naso-pharynx is blocked by the swelling of the erectile tissues covering the turbinates, and almost complete occlusion takes place. The patient is then forced to breathe with the mouth open, and a resulting condition of dryness of the mucous membrane of the mouth and throat and a choking sensation arising from it follow. The natural tendency of an infant or young child is to keep the mouth shut, so that often when the nose is occluded it breathes with great difficulty when asleep, and its face becomes congested and even cyanotic. On forcing the mouth open the symptoms of congestion and cyanosis disappear, and the child begins to snore, and breathes with comparative comfort so long as its mouth remains open, until the dryness of the throat wakes it up.

The prognosis in these cases of acute rhinitis is usually good. The disease runs its course in a variable period of from three days to a week, and, unless the child is subjected to fresh exposure, it recovers entirely. The prognosis, however, as I have already stated, varies in accordance with the age of the individual attacked. The danger that a young debilitated infant may die from exhaustion where the nares are occluded is considerable. You will remember the case which I described to you in a former lecture (page 34), where a puny, ill-cared-for infant died of a simple acute rhinitis. Instances of this kind should warn us that active treatment is indicated.

The treatment should be directed primarily to relieving the nasal occlusion. This is best accomplished by atomizing the nose. In most cases the oil atomizer containing oleum petrolatum album is sufficient to afford relief. In addition to the local treatment, the administration of stimulants where there is exhaustion is indicated. You should also be sure that the infant is taking a sufficient amount of nourishment. This is especially difficult to determine if it is nursing, as under these circumstances it will often hold the nipple in its mouth and apparently suck, while its breathing is so much disturbed by the nasal obstruction that it does not draw much milk from the breast. The various drugs which have been recommended for acute rhinitis have not in my hands proved to be of much use. I have occasionally found that a few drops of the tincture of euphrasia repeated three or four times at intervals of an hour will seemingly lessen the nasal secretion.

As an instance of this class of cases I shall report to you the case of an infant (Case 396) who had an attack of acute rhinitis when she was four months old. Although she was well nourished and fairly strong, yet the occlusion of the nares, which took place



rapidly, produced serious symptoms. She was somewhat cyanotic, refused to take her food, which had to be forced down her throat, and was sleepless, while her strength failed rapidly. She was cared for by a trained nurse night and day, the oil spray was used at frequent intervals, and stimulants were given, with the inhalation of oxygen once every three or four hours. Under this treatment she improved slowly and recovered entirely.

In older children the serious symptoms which I have described do not occur, as a rule, and the disease is not much more significant than the coryza of the adult.

**PURULENT RHINITIS.**—A rather rare form of rhinitis is at times met with in which there have been a number of acute attacks and the process has become somewhat chronic. In these cases the discharge is essentially purulent, and the name purulent rhinitis has therefore been adopted.

This form of rhinitis is not accompanied by any especial enlargement of the turbinated bodies, and narrowing of the nasal passages is not a prominent symptom. The symptoms are chiefly a purulent discharge from the nostrils, and redness and excoriation produced by the acrid character of the discharge.

The prognosis of purulent rhinitis is good, except in extremely debilitated children.

The treatment is the same as in the catarrhal form, especial attention being paid to cleansing the nose with alkaline solutions and thus alleviating the irritation produced by the discharge.

I have a case here in the ward which illustrates the purulent form of rhinitis.

This boy (Case 397) is two and one-half years old. So far as we can ascertain, there has been no especial disease in his parents which would be significant in connection with the present condition of his nose. He is said to have been sick for four weeks. The attack began with fever and general discomfort in connection with the nose. Somewhat later a discharge began to come from the anterior nares and also from the right ear. Up to the time of this attack he had always been healthy and well developed, and is said to have been bright and to have talked as well as is usual for children of this age. During the last two weeks he has grown worse. There has been an increased discharge from the nares. He has become rather dull and apathetic, has lost his appetite, and has stopped speaking. The child lies in bed, or at times gets restless and sits up; his face has a dull expression; he will not speak, and he shows considerable hebetude. There is very little discharge from the ear, but a profuse purulent discharge from both nares. The discharge is evidently irritating, as the upper lip has become excoriated and swollen. He has now had the disease for five weeks. I have detected nothing abnormal in any of the organs except the nose. An examination made yesterday by Dr. Coolidge, one week after the child entered the hospital, showed that there were no adenoid growths or foreign bodies in the nose or naso-pharynx. The pharynx was somewhat congested, but showed no especial pathological condition, and the tonsils were not enlarged. The temperature has varied from 36.6° to 38° C. (98° to 100.5° F.). No cause has been discovered for the attack.

(Subsequent history.) The child, under simple treatment directed to washing out the nose with warm alkaline solutions, and with especial attention to a nourishing diet, improved gradually, and three months from the beginning of the attack was discharged from the hospital cured. The hebetude passed away; he talked as well as ever; he had a good appetite, and a normal temperature; the bowels were regular, and the ears, nose, naso-pharynx, and pharynx were in a normal condition.

In connection with these cases of purulent rhinitis I wish to call your attention to the fact that a purulent discharge from the nose may be the result of an unsuspected foreign body in the nasal passages. This is especially likely to be the case if the discharge is from one side only. It frequently occurs in children, as they are very apt to push various bodies up their noses. If the foreign body happens to be a piece of thin paper or other soft material, it may not cause much nasal obstruction, and its presence may easily be overlooked even when a probe is carefully used in making the examination.

**HYPERTROPHIC RHINITIS.**—This form of rhinitis is rare in infancy and childhood, and I shall therefore merely refer to it. Rhinitis is spoken of as hypertrophic when in addition to a chronic inflammation of the mucous and submucous tissues of the nose there is an actual hypertrophy of the mucous membrane, which results in occlusion of the nares and consequent interference with respiration and the removal of the normal discharges from the nose. One of the most common causes of hypertrophic rhinitis is the occlusion of the posterior nares by adenoid growths, which interfere with the normal nasal secretions by retaining them in the nasal cavity and allowing them to decompose. A recurrent acute rhinitis may also be an etiological factor in hypertrophic rhinitis.

The most marked symptom in hypertrophic rhinitis is the nasal obstruction, which usually alternates from one side of the nose to the other. As would naturally be expected from the lesions, the symptoms are those of great restlessness, especially at night, and various reflex phenomena connected with the throat and the larynx. Thus, there may be continued cough, and, where the Eustachian tubes are occluded, deafness and a resulting hebetude. At times interference with speech results. There is not much nasal secretion in these cases, which aids us in the differential diagnosis from the other forms of rhinitis of which I have just spoken.

The treatment of these cases when they are dependent upon growths in the naso-pharynx is the surgical removal of such growths. Mild astringent sprays should be used, and the oleum petrolatum spray which I have just recommended in catarrhal rhinitis. As a rule, these cases should be placed in the hands of a specialist.

**ATROPHIC RHINITIS (Ozæna).**—By atrophic rhinitis is meant a condition of the nose characterized by atrophy of the mucous membrane and of the bony prominences within the nose, accompanied by what has been termed a dry catarrh, as a result of which the secretion of the nose forms crusts, which undergo decomposition and become fetid. It is also called ozæna. The disease is one which attacks older children rather than infants, and its etiology is obscure. According to Bosworth, it arises from the purulent form of the disease, and he states that as long as the desquamation of epithelium, which is the predominant lesion of purulent rhinitis, is confined to the superficial epithelial cells, the disease is attended with a thick and purulent discharge, but sooner or later the desquamative process extends to the



epithelial lining of the muciparous and follicular glands. The glandular function is thus impaired, and the muco-purulent discharge becomes thick and firmly adherent in the form of crusts to the sinuosities of the nose. This film of desiccated muco-pus in drying contracts the underlying turbinated tissues in such a way as to interfere with the circulation of the blood, a condition which limits glandular action still more and conduces to general atrophy.

The symptoms of atrophic rhinitis are the formation of crusts and the presence of fetor.

Although the tissues which have actually been destroyed by the atrophic process cannot be restored by treatment, the patient can be entirely relieved of the crust formation and fetor by persistent and patient local washing and applications. The details of treatment differ according to the extent and character of the disease. Crusts may be removed by spraying or douching, great care being taken to prevent the washing fluid from entering the Eustachian tubes. If this is not sufficient to remove the crusts, the nasal cavities must be illuminated with a head-mirror, and the crusts carefully brushed off with a cotton-stick. The formation of dry, hard crusts is often prevented by frequent spraying with an oil. Local applications of different substances are of use in many cases, but these should, as a rule, be carried out under the direction of a specialist in the treatment of diseases of the nose.

**MUCOUS POLYPUS.**—This is a pedunculated connective-tissue growth originating from the mucous membrane of the middle turbinate bone. It is rare in children. It does not grow on a healthy mucous membrane, and is always preceded by some morbid condition of the nose. It is often multiple.

The symptoms begin with a nasal discharge followed by nasal occlusion. The diagnosis is easily made by a mirror and a probe. The treatment is the removal of the growth.

**EPISTAXIS (Hemorrhage from the Nose).**—During the period of early childhood hemorrhage from the nose is not uncommon. I have occasionally met with epistaxis in young infants, but in my experience it is rare in the early months of life. In older children recurrent epistaxis, especially if unilateral, points to the presence of an erosion or a varicose condition of the veins in the cartilaginous septum near the external opening of the nose.

Unless the individual happens to be affected by hæmophilia, epistaxis is not especially dangerous, and usually its occurrence ceases as the child grows older.

The application of pressure on the side of the base of the nose and the use of ice are usually sufficient to stop the hemorrhage. If the epistaxis is due to the varicose condition just spoken of, it can be readily controlled temporarily by a plug of cotton pressed upon the bleeding part. For a permanent cure, cauterizing the bleeding part may be necessary.

**NASO-PHARYNX.**—I have described in a previous lecture (page 33) the anatomy of the naso-pharynx. Although this cavity is small and

apparently insignificant, yet it plays a very important part in a number of the diseases to which children are liable. The condition which makes this portion of the respiratory tract especially important is the presence of the pharyngeal tonsil which lines its cavity.

**HYPERTROPHY OF THE PHARYNGEAL TONSIL (Adenoid Growths).—**The glandular or lymph tissue which lines the vault and posterior wall of the naso-pharynx is very similar to that which composes the faucial tonsils, and is called the pharyngeal, third, or Luschka's tonsil, Luschka having first described it. Under certain circumstances this tissue becomes hypertrophied, and gives rise to the condition which is usually designated as adenoid growths.

**ETIOLOGY.**—Hypertrophy of the pharyngeal tonsil, although it may occur in infancy, is uncommon before the second or third year. The disease is essentially one of childhood, as it very seldom develops after puberty. Acute inflammatory conditions or some obstruction in the nose are probably the inciting causes of adenoid growths.

**PATHOLOGY.**—The pathological condition which is found in the lymph tissues of the naso-pharynx is an hypertrophy which is very similar to the hypertrophic condition of the faucial tonsils, except that the latter contain a greater amount of connective fibrous tissue. The hypertrophy may be of greater or less extent, sometimes not being sufficient to cause any especial occlusion and at other times completely occluding the posterior nares.

**SYMPTOMS.**—The first and most prominent symptom which is usually noticed in children who have this disease is that they breathe with their mouths open at night and snore. As the nares become more occluded the child begins to breathe through its mouth also when it is awake. The interference with the proper passage of the air to the larynx and lung results in a chronic form of pharyngitis and laryngitis, while the blocking of the nasal end of the Eustachian tubes may result in a chronic catarrhal condition of the middle ear. Any or all of these symptoms may arise in an individual case according to the amount or position of the obstructions. The child's expression changes, and is almost characteristic when the disease is fully developed. It holds its mouth open, the lower jaw appears to drop, the lips are apt to be thick and expressionless, and when mental dulness is added to the other symptoms it has a stupid look. If this condition continues after the seventh or eighth year, the bridge of the nose is apt to be prominent and its sides to look pinched; the palate may be markedly arched, and the upper jaw narrowed laterally so as to crowd the teeth. The faucial tonsils may or may not be enlarged, but are usually so. This enlargement of the faucial tonsils is, as a rule, secondary to the affection of the pharyngeal tonsil, and not its cause.

**DIAGNOSIS.**—The diagnosis of hypertrophy of the pharyngeal tonsil is not difficult in a marked case or if it is possible to examine the child's naso-pharynx. In young infants the posterior nasal space is so minute that it is almost impossible to reach it. The diagnosis can often be made



simply by the appearance of the child, as there is no other disease which especially simulates this condition. A definite diagnosis, however, can be made only after the hypertrophied tonsil has actually been seen or felt.

I would impress upon you the great importance of learning to detect by means of the finger the presence of an enlarged pharyngeal tonsil. This acquirement is necessary, not only for the purpose of diagnosing the presence of this disease, but also in order to determine correctly the cause of many other abnormal conditions. The examination with the mirror in the throat is usually so difficult in young children that the direct detection by means of the finger is often the most applicable means to employ in these cases. The child should have a blanket pinned around it tightly, so as to keep it from moving its arms. It should be held firmly sitting in the nurse's lap. You can then hold the child's head with one arm, pressing the cheek between the back teeth with the forefinger, then pass the forefinger of the other hand gently, firmly, and quickly over the base of the tongue and behind the soft palate until it reaches the posterior wall of the pharynx. Then, quickly turning the finger upward, you can easily feel whether the cavity of the naso-pharynx is clear or whether it is more or less filled by a soft, spongy mass, the hypertrophied pharyngeal tonsil. There is usually a little blood on the finger when it is withdrawn, as the growth is friable and bleeds easily. This examination is not, as a rule, very painful to the child, but produces a certain amount of discomfort from a choking sensation. When the finger is once in the mouth, it is not wise to take it out again until you have completed your examination, as the child can rarely be induced to allow you to make a second examination. In passing the finger over the base of the tongue you must be careful to get the finger behind the soft palate, and not to push it upward and backward, for in this case the soft tissues of the palate may feel like an adenoid growth. The child can be prevented from biting the finger by simply keeping the cheek pressed between the teeth as I have just described.

Lack of development of the chest with flattening of the front of the thorax may be caused, not, as was formerly supposed, by the enlargement of the faucial tonsils, but by the occlusion caused by the hypertrophy of the pharyngeal tonsil. This hypertrophy with its resulting nasal occlusion may also be the cause of pharyngitis, laryngitis, and perhaps of bronchial catarrh or asthma, which can be cured only by the removal of the primary cause, the pharyngeal tonsil.

In order to impress upon you the chief points in the diagnosis of these cases I show you this little girl.

She (Case 398) is ten years old, and presents a typical picture of this disease.

You see that her mouth is held open and that she evidently has complete occlusion of the posterior nares, the anterior nares on examination being found entirely free. You will notice the pinched look of the face on either side of the nose and the prominence of the bridge of the nose. The child is dull, the dulness having increased as the other symptoms of the adenoid growth have developed, and her face now has a stupid expression. When

you have once seen and studied a case of this kind, you will have no difficulty in making a diagnosis by simple inspection. On examining the child's mouth you see that the palate is very much arched, that the tonsils are enlarged, that the soft palate is slightly pushed forward, and that the pharynx is narrower than normal.

(Subsequent history.) After removal of the adenoid growths and faucial tonsils by Dr. Coolidge she found no difficulty in keeping the mouth closed, not only while awake, but also at night, and slept much more quietly than before. Her general health improved, and the development of her face during the remainder of its growth will undoubtedly be normal.

#### CASE 398.



Hypertrophy of pharyngeal tonsil (adenoid growths). Female, 10 years old.

**PROGNOSIS.**—The prognosis of cases of hypertrophy of the pharyngeal tonsil varies greatly, for there are all forms and degrees of the affection. In some cases the swelling of the lymph-tissues occurs only at intervals when the child has been subjected to exposure in inclement weather; it will then show itself simply by occlusion of the nares, with the resulting discomfort, lasting for some weeks, but disappearing eventually as the weather becomes milder or if the child is taken to a different climate. In most cases, however, where the affection is at all pronounced it becomes chronic, and the symptoms usually increase in severity up to about the time of puberty. You must remember that the naso-pharynx has an important function besides being a passage-way for the air. It lubricates the pharynx, and by the action of its muscles opens the Eustachian tubes during the acts of swallowing and yawning, thus ventilating the ear. You will see, therefore, that the prognosis must vary according to the degree in which any of these functions are interfered with. Where the children become deaf they may gradually lose the power of speech. Again, from being deaf they may fall into a condition of hebétude which sometimes closely simulates idiocy, though it is not true idiocy, for the mental condition quickly changes when the cause of the disturbance has been removed. Where the disease is diagnosed in its early stages, or later, unless irreparable injury has been



done to the ear or the general development, the prognosis is very favorable, provided the proper treatment is carried out.

**TREATMENT.**—The best treatment of these cases is to remove the adenoid growth at once. The operation in the hands of a skilful surgeon is not dangerous, and should be unhesitatingly advised. There are a number of methods which have been employed in operating on these cases. The child should be thoroughly etherized. Some operators prefer to have the child held sitting in the lap of an attendant, others to have it lying down with its head bent backward. The soft palate is drawn forward by means of a palate-hook held in the left hand. A pair of post-nasal forceps held in the right hand is introduced, closed, into the naso-pharyngeal cavity. The blades are then opened, and pieces of the mass are grasped one after the other and pulled off gently: under no circumstances is force to be exerted. With proper care and assistance there is no danger to the child, and often in ten or twenty minutes a morbid condition which has existed for years may be practically cured. There are, of course, many details in this operation which must be thoroughly understood in order that it should be successful. These details, however, need scarcely be mentioned here, as the operation should be performed only by one whose work has especially adapted him for it.

These growths when not extensive are sometimes removed even without ether with the curette or the finger-nail.

I have here a little boy who illustrates the benefit of operative treatment for the removal of the pharyngeal tonsil when hypertrophied.

He (Case 399) is four years old. You see that he has a very bright expression, and he speaks well; he shows nothing abnormal in connection with the shape of his nose or face. He hears well, he sleeps with his mouth shut, and has a free passage of air through a perfectly normal nose and naso-pharynx. When he was three and a half years old it was noticed that he snored at night, breathed with his mouth open, and was subject to continual attacks of rhinitis and naso-pharyngeal occlusion. Following these attacks his hearing became affected, and, while in his second year he had been bright and always ready to play with his parents, he became dull, and did not care to play with others, but would sit for hours playing by himself with his toys.

A digital examination showed a mass of considerable size blocking the posterior nares. On the removal of this mass, which proved to be an hypertrophied pharyngeal tonsil, rapid improvement took place in his general condition, the dulness and hebetude disappeared, and within the last month he has returned to the normal condition which he represented in his second year.

There are other growths which occur in the naso-pharynx, such as sarcomata. They are, however, too rare to need especial description.

**PHARYNX.**—Diseases of the pharynx in children are especially those affecting the tonsils, the uvula, the soft palate, and the posterior wall of the pharynx.

**TONSILLITIS.**—By tonsillitis is meant an inflammation of the tissues of the tonsil. This inflammation may be acute or chronic.

**Acute Tonsillitis.**—Acute tonsillitis may be simply an inflammatory

condition represented by enlargement and reddening, *simple tonsillitis*, or the inflammation may be especially located in the crypts of the tonsil, in which case it is commonly designated *follicular tonsillitis*.

The other affections of the tonsils, such as occur in the course of the exanthemata and in diphtheria, constituting the pseudo-membranous form of the disease, can best be described in connection with the especial diseases in which they arise, and I shall therefore speak only of the two forms to which I have just alluded.

*Acute Simple Tonsillitis*.—It is probable that the cause of the acute simple form of tonsillitis is a microbe. The child is usually attacked suddenly, with a heightened temperature, 38.8° to 39.4° C. (102° to 103° F.), fever, restlessness, and sometimes vomiting and loss of appetite. Young children do not complain of the throat so much where the tonsils are affected as do older children and adults. In fact, in many cases, unless the throat is actually inspected, it would seem as though it were not a local affection of the throat, but some general disease affecting other parts of the system. You should be especially on your guard, therefore, not to have your attention diverted from the throat, but under all circumstances where these symptoms arise in young children, even though they apparently swallow without discomfort, to examine the throat before deciding whether or not some other disease is developing.

On inspection of the throat the tonsils are seen to be enlarged in different degrees and to be of a uniform bright red color.

The mucous membrane of the pharynx is, as a rule, much reddened; the soft palate may also be reddened, but not necessarily. The symptoms continue for a day or two and then diminish, and the child usually recovers in about a week.

The local application of a cleansing spray, and the administration of ice if desired to relieve the discomfort, constitute all that is necessary for the treatment of these cases. It is best not to disturb the mucous membrane with applications on swabs or brushes.

*Acute Follicular Tonsillitis*.—In the acute inflammation of the tonsil which is usually called follicular tonsillitis, in addition to the general inflammation of the tonsils the crypts are especially affected. The cause of this form of tonsillitis is undoubtedly infection by some pathogenic germ. It is probable that more than one form of germ is capable of causing it. Many of the pathogenic germs which infest the mouth or the throat may be found in the crypts in this disease, but the especial germ by which we can characterize the disease has not yet been determined.

**SYMPTOMS**.—As a rule, the disease is characterized by an acute onset, with a heightened temperature, 39.4° to 40° C. (103° to 104° F.), loss of appetite, and general malaise. I have often noticed, however, that the symptoms of a marked follicular tonsillitis are not so acute and do not so definitely point to the throat in young children as they commonly do in older children and in adults. On examining the throat the tonsils are seen to be



enlarged, reddened, and in the early hours of the disease to show a little swelling of the orifices of the crypts, as though a secretion within them was about to burst the overlying mucous membrane and appear on the free surface. Later this actually occurs, and the tonsils are seen to be studded with white or grayish-white spots. These do not appear on the soft palate or uvula, though they may appear on the base of the tongue and the posterior pharyngeal walls. The mucous membrane of the pillars of the palate, of the uvula, and of the soft palate are usually reddened, and there is very apt to be decided reddening and even swelling of the mucous membrane and follicles of the pharynx. As the disease progresses these spots may coalesce and, adhering to the surface of the tonsil, form a pseudo-membrane which is often impossible to distinguish from diphtheria without a bacteriological examination. As there is a direct connection between the tonsils and the cervical glands, the latter are liable to be involved, though any great swelling of the cervical glands in connection with acute tonsillitis is uncommon.

The disease is self-limited, and runs its course in two or three days or a week, at the end of which time the general symptoms subside, the appetite returns, the temperature becomes normal, and the child, although it is left somewhat weakened by the disease, seems as well as ever. The tonsils themselves, however, do not for some time regain their original size, and the exudation often remains in the crypts and may cause a chronic irritation with a tendency to recurrence.

DIAGNOSIS.—The differential diagnosis of follicular tonsillitis is to be made from the various forms of stomatitis, which I have already sufficiently described, and from diphtheria, of which I shall presently speak. It is now very generally known that it is impossible absolutely to exclude diphtheria by the morbid appearances seen on the tonsils. In the great majority of instances, however, where the attack is acute, where the cervical glands are not especially involved, where the white spots on the tonsils are clearly located in the orifices of the crypts, and where there is no appearance of a membrane on the uvula or the soft palate, we can make the clinical diagnosis of follicular tonsillitis with considerable certainty, but never surely without a bacteriological examination.

PROGNOSIS.—The prognosis of follicular tonsillitis is in almost every case favorable, and is rendered unfavorable only by the complication of tonsillar abscess. But you must remember that in an inflamed tonsil pathogenic organisms, such as those of diphtheria, are more apt to develop.

TREATMENT.—The treatment of acute follicular tonsillitis, according to my experience, should be entirely symptomatic. It is a self-limited disease, and in a vast majority of cases is not benefited by the administration of any drug internally or by local applications. In order to avoid the invasion of the various pathogenic germs during the progress of the tonsillitis I am in the habit of having the throat kept thoroughly clean with mild solutions of chlorate of potassium or borate of sodium. Holding pieces of cracked ice in the mouth often affords considerable relief. In young children, as a rule,

I make no local application beyond allowing them to swallow cold solutions of chlorate of potassium in the strength which I have already advised (page 783). Small doses of quinine according to the age of the child are indicated where there is much exhaustion or malaise following the attack.

I happen to have here in the wards a case of acute follicular tonsillitis.

This little girl (Case 400, Plate VIII., facing page 781, Follicular Tonsillitis), four years old, has an attack of follicular tonsillitis and illustrates what I have just told you.

She was taken sick two days ago with a heightened temperature of about  $39.4^{\circ}$  C. ( $103^{\circ}$  F.), loss of appetite, and general malaise. She did not complain of her throat, and swallowed without difficulty. Nothing abnormal was found in any of the other organs, but on inspecting the throat the tonsils were seen to be enlarged and much reddened, and one or two of the orifices of the crypts were somewhat raised above the general surface of the tonsil. On the following day a number of white spots of different sizes appeared on both tonsils. To-day you see that the redness is mostly confined to the tonsils, and affects the uvula and palate very little. On the inner surface of both tonsils the exudation has coalesced, so that it has an appearance very much like that of a pseudo-membrane. It is not uncommon in follicular tonsillitis for this coalescence of the exudation to take place on the surface of the tonsil which points towards the median line of the throat. The other appearances of the tonsils are characteristic of follicular tonsillitis. On the upper left-hand corner of the left tonsil, close to the arch of the palate, you will see an enlarged cryptic orifice which has not quite broken down, and which appears as a light red prominence on the general surface of the tonsils. The orifices have a like appearance in various parts of both tonsils. On the anterior surface of the left tonsil are two white spots, caused by the exudation from the crypts. In the upper part of the right tonsil are three smaller yellowish-white spots, and lower down on the tonsil a grayish-white rather large spot, all of them due to the same cause. There are no other lesions in the throat, and the cervical glands are not involved.

Cultures made from this exudation did not show the presence of the Klebs-Loeffler bacillus.

In a case of this kind, with appearances such as you see in this throat, you can say that the disease is probably follicular tonsillitis and not diphtheria, especially when the absence of the Klebs-Loeffler bacillus has been proved.

The child now has a normal temperature, and is improving rapidly. In a few days it will be entirely well. The lesions, as you see, are still present in the throat, but the disease has run its course and has ceased to produce any general symptoms.

The treatment has been simply to feed the child from time to time with small doses of iced milk. No local applications and no drugs have been used.

**Chronic Tonsillitis.**—After an acute tonsillitis has recurred a number of times, or where a chronic form of inflammation has affected the tonsil from the beginning, an enlargement of the tonsils takes place, which consists of an hypertrophy of their tissues. This is what is known as hypertrophy of the tonsils.

Although this hypertrophy of the faucial tonsils may exist without a corresponding affection of the pharyngeal tonsil, yet it is very apt to be secondary to this latter condition.

**PATHOLOGY.**—The terms chronic tonsillitis and hypertrophied or enlarged tonsils are commonly used to express the same condition, especially in children, in whom chronic inflammation of the tonsils unaccompanied by enlargement seldom occurs. This enlargement is always due, at least in



part, to true hypertrophy, generally accompanied by more or less inflammatory deposit. If the parenchymatous or glandular tissues are especially affected we find a soft, more or less red and vascular tonsil, with large crypts, often containing much secretion. In the interstitial form the tonsil is hard and tough, the crypts less prominent or even very small, and the vascularity much diminished. These types are the two extremes; in most cases the enlargement is essentially one of hypertrophy. The tonsils may be only moderately enlarged, or their size may be so increased that they meet, touching each other in the median line. The growth is usually towards the median line. In examining a child for enlargement of the tonsils care must be taken that the pharynx is not contracted by gagging at the time the examination is made. The act of gagging, which is easily brought about in children by a careless use of the tongue-depressor, brings the tonsils towards the median line, thus giving tonsils of normal size the appearance of being large and obstructive.

SYMPTOMS.—The symptoms of hypertrophy of the faucial tonsils vary according to the degree of enlargement. Normally the tonsils can scarcely be seen on inspection of the throat. When only moderately enlarged they may produce no symptoms whatever beyond a feeling at times of slight irritation in the throat. When in this condition, however, they are more apt to be irritated by various external influences and to be the source of recurrent acute affections of the throat. When considerably enlarged they may still not produce any marked symptoms, provided that the passage of air through the naso-pharynx is unobstructed. They may, however, even when the pharyngeal tonsil is not enlarged, cause obstruction in the naso-pharynx by pressure as they enlarge upward and backward. When this happens, the same interference with the breathing and development of the child takes place as when the obstruction is primarily in the naso-pharynx. These symptoms are the same as I have already described in speaking of hypertrophy of the pharyngeal tonsil, and therefore need not be detailed here.

Occasionally difficulty in swallowing and thickness of speech may arise where only the faucial tonsils are enlarged.

PROGNOSIS.—The prognosis in cases of hypertrophy of the faucial tonsils depends upon these varied anatomical conditions which I have just explained to you. So long as the tonsils do not encroach on the naso-pharynx the prognosis, so far as injury to the child is concerned, is good. You must always remember, however, that the enlargement of the tonsils is a fertile source of irritation which may prepare the way for serious disease produced by the various micro-organisms. The prognosis as to their disappearing is not especially good, as they seldom recover their normal size without active treatment when once hypertrophied, though they generally diminish slowly in size after puberty.

TREATMENT.—Local applications for the reduction of hypertrophied tonsils are useless. Some success has been obtained by Gampert by what is called discission of the tonsils. Leland has strongly advocated this treat-

ment, which consists in making slits in the tonsil with a knife especially devised by him.

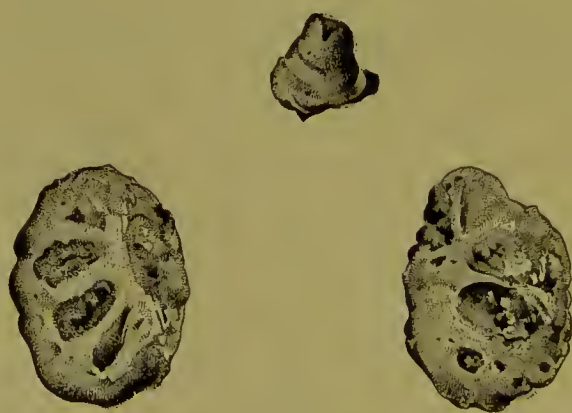
The most thorough and certain way of curing the disease is, however, by excision. This should be done with the tonsillotome, and it is best to etherize the child for the operation. It is considered wiser to amputate the tonsil than to enucleate it. After the operation the child should be made to gargle with a solution of borate of sodium for two or three days, and should be given only milk for its food.

As a result of attacks of acute inflammation the hypertrophied tonsils may have become adherent to the anterior or the posterior pillars of the fauces to such an extent that the guillotine either cannot be used, or not without danger of wounding these pillars. In such cases it may be necessary to revert to dissection or to the galvano-cautery to remove the tonsillar tissue.

I have an interesting case of hypertrophy of the faucial tonsils and of the pharyngeal tonsil to show you to-day.

The child (Case 401), a boy, seven years old, has for the past two years been under my observation. He was a healthy infant and well developed until his fifth year, when he did not continue to grow normally and lost much in weight. He became rather dull, and although he did not have any especially severe symptoms, yet he frequently had mild attacks of rhinitis, pharyngitis, and tonsillitis. After a number of recurrent attacks of tonsillitis the tonsils remained enlarged, and for the past two years he has presented in his throat the condition of hypertrophied tonsils. Various applications have been made to the tonsils without any favorable result. The specialist who first saw the child told the parents that the tonsils would probably decrease in size as the child grew older, and that operative treatment would not be advisable. The child is now beginning to breathe with his mouth open at night, and to be very restless when asleep, and has become very nervous. The chest, as you see, is decidedly flattened, and the sides of the nose are beginning to look a little pinched. The child is evidently suffering from obstruction to the entrance of air to the lungs, and I have therefore decided that operative treatment is indicated.

FIG. 101.



Part of the hypertrophied pharyngeal tonsil in the upper part of the figure. Excised surfaces of the faucial tonsils in the lower part of the figure. Male, 7 years old.

Dr. Farlow will now examine the case before you and operate on it. On passing the finger up into the naso-pharynx it is found that the pharyngeal tonsil is markedly enlarged, and as the result of the obstruction the breathing of the child is interfered with, and also its development.



The child is now etherized, and Dr. Farlow has first, as you see, excised both tonsils with the tonsillotome and has then removed the pharyngeal tonsil with the forceps.

Here you will see the crypts and the intervening connective tissue of the excised faucial tonsils, and also the soft spongy tissue of the pharyngeal tonsil. (Fig. 101, p. 814.)

(Subsequent history.) Within six months after the removal of the pharyngeal and faucial tonsils the boy presented an entirely different appearance from what he did before the operation. His chest had developed, he had a better color and a good appetite, he had ceased to be nervous, slept with his mouth closed, and showed no tendency to the recurrence of the rhinitis from which he formerly suffered continually.

**PERITONSILLAR ABSCESS.**—In some cases an inflammatory process resulting in suppuration occurs in the cellular tissue around, above, or behind the tonsil, constituting an abscess which is called peritonsillar. It is rather rare in early childhood. The disease is usually preceded by a certain degree of simple tonsillitis, and when it develops the temperature rises, perhaps to  $40^{\circ}$  or  $40.5^{\circ}$  C. ( $104^{\circ}$  or  $105^{\circ}$  F.), and the child evidently suffers much pain.

On inspecting the throat in these cases a unilateral swelling is seen in the neighborhood of the tonsil, as a rule, pushing the soft palate forward, and the tonsil towards the median line.

The prognosis is in almost every case favorable, except those which have been neglected or improperly treated. There is sometimes extensive burrowing of the pus, and hemorrhage, or even œdema of the larynx, may occur.

The treatment is to locate the abscess by careful palpation and to open it under strict antiseptic precautions with a guarded bistoury.

**PHARYNGITIS.**—An inflammatory condition of the posterior wall of the pharynx is rather rare in infancy, but is not uncommon in children. It is usually coincident with an inflammatory condition of the naso-pharynx or of the tonsils, but in a certain number of cases it is so much more pronounced in the posterior wall than elsewhere that it can be described as a separate disease. The diseases of the posterior wall of the pharynx are either (1) a simple catarrhal condition of the mucous membrane or (2) an inflammatory process in which the follicles are especially affected. In addition to these conditions, pus may form behind the mucous membrane, producing a retro-pharyngeal abscess. The inflammatory lesions of the pharynx may be acute or chronic.

The conditions which give rise to pharyngitis are the same various morbid processes that involve the parts in the neighborhood of the pharynx, such as the naso-pharynx and the tonsils. These causes I have already mentioned. There also seems to be a connection between certain irritations arising in the gastro-enteric tract and the pharynx. Where this occurs it is usually the chronic form of pharyngitis which is met with, and the causal connection between these two distant parts of the economy is probably of a reflex nature.

**Acute Simple Pharyngitis.**—The pathological condition which is found in the simple acute form of pharyngitis is an acute inflammation

characterized by a slightly heightened temperature, a hyperæmic condition of the blood-vessels of the posterior wall of the pharynx, and a certain amount of swelling and of serous exudation.

The symptoms are discomfort in swallowing, and at first a feeling of dryness in the throat, followed later by an exudation of viscid mucus. The child does not seem especially sick with this disease, and the length of the attack varies according to the influences which are causing it.

**Acute Follicular Pharyngitis.**—The acute follicular form of pharyngitis does not differ materially in its symptoms from the simple form, and in fact both forms are so frequently combined that a clinical distinction need scarcely be made between them. On examining the pharynx in this form of pharyngitis, in addition to the appearances which are seen in the simple form, the follicles will be found enlarged and raised above the surface of the mucous membrane.

**TREATMENT.**—The treatment of both forms of pharyngitis is essentially local, and is, as a rule, by applications to the inflamed mucous membrane in the form of a spray, either directly, or indirectly through the nose. The spray is essentially for the purpose of cleansing and thus soothing the inflamed mucous membrane, and should consist of mild alkaline solutions such as this one (Prescription 76):

PRESCRIPTION 76.					
<i>Metric.</i>			<i>Apothecary.</i>		
		Gramma.			
℞ Sodii chloridi . . . . .	0	3	℞ Sodii chloridi . . . . .	gr. v;	
Sodii bicarb.,			Sodii bicarb.,		
Sodii boratis . . . . .	āā	0 9	Sodii boratis . . . . .	āā gr. xv;	
Aquæ rosæ . . . . .	30	0	Aquæ rosæ . . . . .	℥i;	
Aquæ . . . . .	90	0	Aquæ . . . . .	℥ij.	
M.			M.		

Sig.—Spray for pharyngitis.

The chronic form of pharyngitis is usually accompanied by an irritating cough, which is most pronounced at night and in the morning. You should not think that these children who are coughing continuously and often losing in weight and looking weak and anæmic are necessarily affected with bronchitis. This series of symptoms is frequently looked upon as diagnostic of bronchitis, when it really arises from pharyngitis, and can be cured by treating the latter disease.

The treatment of chronic pharyngitis is to remove any hypertrophic condition of the tonsils, tongue, or nose, to regulate carefully the child's general nutrition, and to avoid undue exposure to inclement weather or to air vitiated in any way, as by arsenic or dust. Local applications of a one per cent. solution of nitrate of silver, followed immediately by thorough cleansing with sterilized water, are sometimes indicated in the more intractable cases.

**Elongation of the Uvula.**—Accompanying pharyngitis, usually in its chronic form, an elongation of the uvula is at times met with in children.



This condition may arise from a relaxed condition of the muscles of the soft palate and of the uvula, or may consist simply of a redundancy of the mucous membrane at the tip of the uvula. The general irritated condition of the uvula and the tickling sensation produced by its elongated tip touching the base of the tongue cause a harassing cough, which by its persistence weakens the child, gives rise to loss of appetite, and interferes with its nutrition.

Local applications of astringents, such as alum, are at times sufficient to restore the uvula to its normal condition, but the disease can be cured quickly by excising the end of the uvula with blunt-pointed scissors. The amputation of the entire uvula is to be avoided, as it has been known to weaken the muscular action of the soft palate.

A papillomatous growth is sometimes found attached to the tip of the uvula or to its side, which causes the same symptoms as elongation of the uvula. The treatment is excision, after which it does not recur.

**RETROPHARYNGEAL ABSCESS.**—Retropharyngeal abscess is a disease which occurs usually during the first year of life and is very rare after this time. The disease may be secondary to injuries of the pharynx, to abscess in the neck, and to disease of the cervical vertebræ, or it may be metastatic from septic processes such as occur in diphtheria. In a certain number of cases it is idiopathic so far as we know.

**PATHOLOGY.**—The pathology of the disease consists in the formation of an abscess in the tissues of the posterior wall of the pharynx, and is more apt to be on one side of the pharynx than in the median line.

**SYMPTOMS.**—The symptoms, whether the disease is primary or secondary, are very much the same. The first symptom is generally difficulty in swallowing, which may go on to entire inability to swallow. The infant is next noticed to breathe in a peculiar way. It holds its head back and its mouth open. The breathing may be described as snorting, and at times as almost stertorous, differing markedly from the whistling sound which is heard in obstruction of the larynx. On examining the throat the soft palate is seen to be pushed forward and to be somewhat anæmic. The posterior wall of the pharynx is bulging, usually unilaterally, is reddened, swollen, tense, and as the disease progresses is found to be fluctuating. In some cases the abscess burrows into the tissues of the neck and appears as a pear-shaped tumor behind the ear. I have met with two cases of this variety where the pus could be reached easily by an external incision.

**DIAGNOSIS.**—The diagnosis must be made chiefly from peritonsillar abscess. This is, as a rule, not difficult unless the latter condition has proceeded so far that the pus by burrowing has invaded the walls of the pharynx. It is usually not difficult to determine the situation of the abscess by passing the finger directly through the mouth to the posterior wall of the pharynx. If there is pus in the tissues of the pharynx a sense of fluctuation will be obtained. The position of the child in cases of retropharyngeal abscess is also significant, and is not that which is

assumed in peritonsillar abscess. It holds its head back, in order to allow a free passage for the air through the occluded pharynx into the larynx.

The diagnosis must also be made from œdema of the glottis, but this is not, as a rule, difficult, for inspection shows that in the latter disease bulging, redness, swelling, and fluctuation of the posterior wall of the pharynx are not present. The characteristic position of the head, also, is not seen in œdema of the larynx.

**PROGNOSIS.**—The prognosis in the cases where the abscess is secondary varies according to the nature of the disease which causes it. Thus, it is an exceedingly dangerous complication in diphtheria, and is one of serious import in cervical spondylitis. In those cases of undetermined origin which are spoken of as idiopathic the prognosis is very good if the proper treatment is carried out at once. We must, however, consider the possibility of the abscess bursting suddenly, the child suffocating by inspiration of pus into the larynx. This has been known to occur where the disease has been left untreated.

**TREATMENT.**—In the idiopathic cases the abscess should be opened at once. The method which I have found satisfactory in the cases which have come under my observation is to have an assistant hold the infant sitting upright in the lap, with a blanket tightly pinned around it so as to prevent it from moving its arms. Another assistant should hold the head. The mouth should then be opened, a guarded bistoury should be introduced into the pharynx and the abscess punctured. As soon as the opening has been made the bistoury should be removed quickly, and the infant's head should be immediately thrown forward and downward, so that the pus will be discharged from its mouth and not inspired into the larynx or swallowed. It is usually necessary after the operation to introduce the finger into the pharynx and to press the walls of the abscess, so as to empty any pus which may continue to collect there and also to keep the opening free. With this treatment, unless some complication should arise, the disease is usually cured in about a week.

Some operators prefer having the child placed in what is known as Rose's position, on its back with the head hanging over the end of the table.

Here is an infant (Case 402), seven months old, who has just been brought to the hospital.

Without examining the throat, you can almost diagnose a case of retropharyngeal abscess by the character of the breathing, which is snorting and labored. You will notice the very characteristic appearance produced by the obstruction in the pharynx. You see that the head is held back and the mouth open; that the infant's eyes are somewhat rolled upward, but that it is perfectly conscious. Its face is slightly cyanotic. On depressing the lower jaw and tongue you see that the soft palate is pressed forward, that its blood-vessels are almost empty, and that it is very pale. On throwing a strong light from the mirror on the posterior wall of the pharynx you see on the left of the median line a swollen, congested, bulging condition of the mucous membrane. On touching the most prominent part of the swelling with the finger you get a sense of fluctuation.

The infant is said to have been sick for two weeks with a cold in its head. Three days ago it began to breathe in this manner and to hold its head back rigidly and somewhat to



the left. It also began to hold its mouth open. It was able to nurse only a few seconds at a time, when it would let go of the nipple and refuse to take it again. It has been growing very weak from lack of nourishment and from the exhaustion arising from the difficulty with its breathing.

## CASE 402.



Retropharyngeal abscess. Male, 7 months old.

(Subsequent history.) An opening in the abscess was made by Dr. Burrell, and a large amount of pus was evacuated. An hour later the child began to choke, and it seemed as though tracheotomy would have to be performed, but pressure with the finger on the walls of the abscess from time to time, surrounding the infant with an atmosphere containing steam, and free stimulation, proved eventually to be all that was necessary for its recovery.

I shall now report to you a case (Case 403) of retropharyngeal abscess which came under my observation some years ago.

An infant thirteen months old, and always strong and healthy, had an attack of acute rhinitis for several days. The rhinitis apparently caused considerable swelling and occlusion of the nares, and the infant after four or five days began to hold its mouth open when breathing and to have difficulty in swallowing. This difficulty in deglutition increased, and it was then noticed that her head was held back. On examining the throat a tense fluctuating swelling was detected in the posterior wall of the pharynx very nearly in the median line. This swelling was incised by Dr. Hooper. A considerable amount of pus was evacuated, and the infant immediately began to breathe more easily and was able to swallow without difficulty. During the next twenty-four hours the abscess filled with pus a number of times, and the pus had to be emptied by pressure with the finger. The infant made a perfect recovery, and has had no return of the disease.

Cases such as this lead me to say a few words upon another method of drainage that I did not speak of when telling you about opening these abscesses that are found in the mouth. In the ordinary case, when the pus has come from the breaking down of glandular material in the posterior wall of the pharynx and already contains pyogenic bacteria, the dangers from reinfection from a wound in the mouth are not serious. But when the pus has come from a tuberculous focus in the cervical vertebræ and contains no other organisms than the bacilli of tuberculosis, and is moreover in direct communication with an active pathological process in the bone, the risks of a secondary septic infection are considerable. It is, therefore, the practice of many surgeons, despite the difficulties of the operation, to attempt to reach the abscess by a careful dissection from the outside of the neck, as it is far easier to keep the wound in that situation aseptic. If there is any sign of the abscess pointing externally, the outside operation should always be preferred.

I happen to have here another case of retropharyngeal abscess to show you, in which the condition is secondary to cervical spondylitis.

CASE 404.



Retropharyngeal abscess secondary to cervical spondylitis.

This child (Case 404) was being treated for cervical spondylitis by Dr. Bradford, when in addition to the drawing back of its head, which you see, it began to have increased difficulty in swallowing and to breathe with its mouth open.

On examining the pharynx a bulging, tense, fluctuating abscess of moderate size was detected.



## LECTURE XLII.

## DIPHTHERIA.

DIPHTHERIA is an acute, highly infectious disease, due to the Klebs-Loeffler bacillus. It is primarily a local affection, the constitutional symptoms being due to the absorption of toxins.

There is frequently a concurrent infection with pyogenic cocci. These organisms produce the secondary inflammations occurring in the disease, and also, by their toxins, give rise to additional constitutional symptoms.

ETIOLOGY.—The Klebs-Loeffler bacillus, first described by Klebs and later more fully identified by Loeffler, is a small organism,  $2.5\ \mu$  to  $3\ \mu$  in length and  $0.5\ \mu$  to  $0.8\ \mu$  in thickness. Its most striking features morphologically are its variation in form and the irregularity in its manner of staining. The ends of the organism are frequently clubbed, and in most cases, when it is stained, it shows a series of clear spaces with intensely stained particles. The form and size vary greatly under various circumstances. I have here a specimen (Fig. 102) from a blood-serum culture from the throat of a child in the diphtheria wards of the Boston City Hospital, which shows the morphology of this bacillus in its typical form.

FIG. 102.



The bacillus of diphtheria.

It grows readily on a variety of culture media, and most readily on the modified blood-serum first introduced by Loeffler. It does not form spores. Welch and Abbot have shown that in fluids it may be killed by an exposure of ten minutes to a temperature of  $58^{\circ}\text{C}$ . ( $136.4^{\circ}\text{F}$ ). Under favorable conditions it may remain alive for weeks, or even months, in fragments of dried membrane. The bacillus of diphtheria is best stained with Loeffler's alkaline methylen-blue solution.

The pyogenic cocci most frequently found in the concurrent infections are the streptococcus pyogenes, alone or associated with the staphylococcus

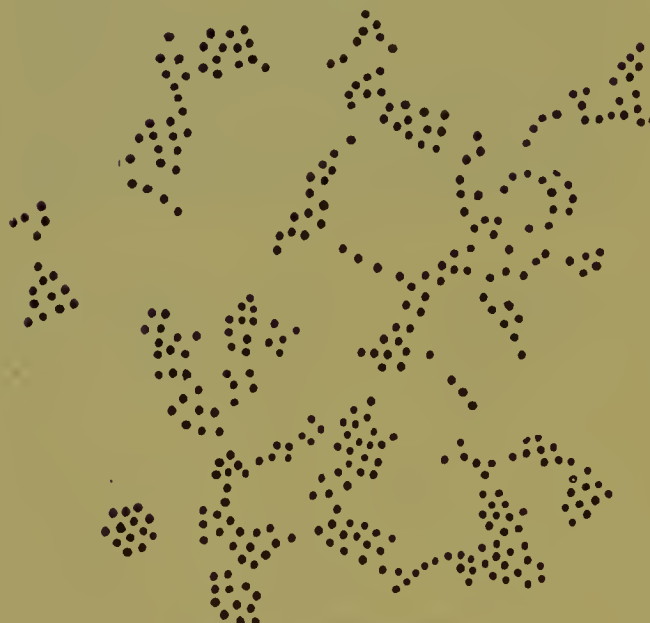
pyogenes aureus, the former being the more important in its results. I have here some specimens (Figs. 103, 104) of these organisms, showing their morphology.

FIG. 103.



Streptococcus pyogenes.

FIG. 104.



Staphylococcus pyogenes.

You will notice that they both appear as dots, the streptococcus showing a tendency to arrange itself in chains, while the staphylococcus is irregularly bunched.

There is no true diphtheria where the Klebs-Loeffler bacillus is not present, but its presence in a healthy throat does not constitute the disease diphtheria, although the individual may be the source of infection to others.

The contagium of diphtheria is contained chiefly in the secretions of the throat and nose, and is communicated usually by direct or indirect contact, and, as a rule, not by the air.

An unhealthy condition of the mouth, nose, or throat predisposes to the



disease, as a lesion of the mucous membrane is necessary for its entrance. Sewer-gas and confined, impure air of any kind may act by weakening resistance to the bacillus, or, by producing a benign lesion in the throat, may offer a suitable nidus for the invasion of the bacillus. Although clinically it has been supposed that animals have primary diphtheria, this has not been conclusively proved bacteriologically.

Although diphtheria may occur at any age, it is rarely met with in early infancy. It is most commonly seen from the second or third to the fifth or sixth year. It may occur more than once in the same individual.

**PATHOLOGY.**—The most constant lesion in diphtheria is the presence of a pseudo-membrane in the upper air-passages, due to a combination of inflammation and coagulation necrosis.

It must be remembered, however, that the same anatomical condition may be caused by other bacteria and by irritants, and also that the process may be simply a catarrhal inflammation which does not go on to the formation of a pseudo-membrane. It is evident, therefore, that there is no pathological condition characteristic of the action of the Klebs-Loeffler bacillus.

The adjacent lymph-nodes are apt to be swollen, and on microscopical examination they often show small foci of cell-necrosis; similar smaller necrotic foci may be found in other parts of the economy, such as the liver and the kidney, and are due to toxic absorption. There is also a general lymphatic hyperplasia, which is relatively greatest in the abdomen. The kidneys ordinarily show only parenchymatous degeneration, but in a few cases of concurrent infection they may present acute lesions. Hemorrhages into the serous membranes are often met with, and the organs in general show degenerative changes due to toxic absorption. Endocarditis is rarely seen. Catarrhal bronchitis and broncho-pneumonia frequently complicate diphtheria, and are caused by the inspiration of the pyogenic cocci. This was demonstrated by Prudden and Northrup in a very able paper which appeared in 1889.

**INCUBATION.**—The time which elapses after exposure to the infection until the first symptoms develop may be only twenty-four hours or may be two or three days. This period, however, is a very indefinite one, since the interval between the access of bacteria to the mucous membrane and the time when they invade the membrane with their toxic effects depends upon whether the tissues of the mucous membrane are vulnerable. Thus, it is probable that the bacillus diphtheriæ may exist in the mouth for an indefinite time without infecting the individual.

**SYMPTOMS.**—The prodromal symptoms of diphtheria are not especially characteristic. They may be acute in character or very mild and of a sub-acute variety. There are apt to be a sensation of chilliness, some heightening of the temperature, and more or less pain in the back and limbs. There is nothing, however, to distinguish this stage of the disease from many of the other affections of children, such as a simple tonsillitis. The child may often complain of discomfort on swallowing, and on examining the throat the

fauces are found to be reddened. In a short time, however, more characteristic appearances will be found in the throat. A pseudo-membrane, white or grayish white, and commonly appearing on the tonsils first, develops, and on the second or third day usually extends to the soft palate and uvula. It may also appear in the pharynx. During this stage the throat becomes much swollen and the tonsils considerably enlarged, sometimes so as almost to meet in the median line. The membrane is usually firmly adherent to the mucous membrane, and as the case progresses it assumes a brownish-gray or yellowish-gray color. In addition to these lesions in the throat, the cervical glands are usually involved and become considerably swollen. The child, as a rule, shows grave constitutional symptoms and loses its appetite. The temperature in diphtheria is not characteristic, and is usually not especially high,  $38.3^{\circ}$  or  $38.8^{\circ}$  C. ( $101^{\circ}$  or  $102^{\circ}$  F.). The pulse is somewhat increased in rapidity, and is weak in proportion to the severity of the disease. In cases of a mild type the symptoms abate towards the end of the first week, the pseudo-membrane separates, leaving a raw surface behind it, the throat becomes less swollen, and the child feels much better. It is, however, usually left much prostrated for a number of weeks, and even in these mild cases the toxic effects of the disease may show themselves in the form of a neuritis, with its accompanying paralysis, many weeks after the diphtheria itself has run its course. There may also even in mild cases be a slight discharge from the nares, owing to the involvement of the posterior nares, and a slight albuminuria.

I have brought you into the diphtheria ward to-day to show you one of these mild cases of diphtheria.

This boy (Case 405), five years old, has been sick for four days. His pulse is somewhat rapid, but of good strength. His respirations are slightly increased, but there is no retraction. There is a slight discharge from the nose, and the cervical glands are somewhat enlarged. He takes his nourishment well, and is in a very fair condition. A culture made on Loeffler's blood-serum of a shred of membrane taken from the throat showed the presence of the Klebs-Loeffler bacillus and a large number of streptococci. The urine contains a small amount of albumin.

I show you this case as especially illustrating the typical appearances of diphtheria in the throat, and in order that you may compare it with the typical appearances of the throat in follicular tonsillitis, which I showed you in a previous lecture (page 781).

On examining this boy's throat (Plate VIII., facing page 781, Diphtheria) you will see small patches of grayish-white pseudo-membrane on the upper part of the left tonsil and spreading to the left arch of the soft palate. The membrane has also involved the right side of the uvula, the right arch of the soft palate, and the side of the right tonsil pointing towards the median line. There is also a patch on the right tonsil and one on the posterior wall of the pharynx. The tonsils are moderately enlarged and reddened, and the mucous membrane of the soft palate is also considerably reddened.

When lesions of this character and having this distribution are seen in the throat you need have no doubt regarding the clinical diagnosis of diphtheria, and should at once have a bacteriological examination made.

**VARIATIONS IN TYPE.**—There are a number of variations which occur both in the severity of the disease and in the locality which is at first attacked or principally invaded.



In some epidemics the Klebs-Loeffler bacillus seems to be far more virulent than in others, and in some individuals it produces much more serious symptoms than in others. The severity of the attack does not always depend upon the extent of the pseudo-membrane. In general the severity of the cases depends on three factors: (1) the virulence of the bacteria, (2) the local resistance, and (3) the general resistance. A number of what may be called atypical cases have been observed and carefully studied, especially by Koplik, where no pseudo-membrane was detected and where the morbid appearances in the throat were those of a simple catarrh or follicular tonsillitis. The virulent Klebs-Loeffler bacillus was detected in these cases, and other children infected by them presented the typical local lesions of diphtheria.

In addition to these mild cases, the Klebs-Loeffler bacillus at times produces a most malignant form of diphtheria. In these cases the child either shows a fairly mild form of the disease for a few days and then suddenly develops the severe form, or it may be attacked at once by very severe symptoms. It becomes dull; the temperature is either slightly raised or may rise to  $39.4^{\circ}$  or  $40^{\circ}$  C. ( $103^{\circ}$  or  $104^{\circ}$  F.), or higher; the pseudo-membrane spreads rapidly; there may be a dusky efflorescence on the skin, simulating closely that which I have described in the malignant form of scarlet fever. There may also be a purpuric condition of the skin. The picture of these septic cases is very characteristic. There is a peculiar, sweetish odor of the breath. There are cyanosis and a marked waxy pallor. There are hemorrhages from the throat and nose, with a profuse mucopurulent discharge from the latter. The cervical glands are often enormously enlarged. The membrane has been known to extend in all directions, and sometimes even through the Eustachian tubes to the external ears. All degrees of severity are met with between the mild and malignant types of diphtheria. The membrane, instead of extending upward to the nasopharynx, as occurs in the malignant cases just spoken of, may spread downward, attacking the epiglottis and the larynx, and cause serious obstruction.

I have already told you that the pseudo-membrane most commonly appears first on the tonsils, thence spreading to the soft palate and to the uvula. The disease may, however, begin in the mucous membrane of any part of the mouth, nose, or throat.

**The Nose.**—Diphtheria sometimes begins in the nose and spreads no farther. In these cases the disease is usually of a mild type, but it is infectious. These cases are especially liable to be overlooked, as the child for one or two days may show merely the symptoms of fever, malaise, loss of appetite, and a discharge from the nose. On examining the nose carefully, however, a pseudo-membrane will often be found. It is, therefore, very important in cases of this kind to have a bacteriological examination made, and to isolate the child until it is determined that the Klebs-Loeffler bacillus is not present. These cases are probably a prolific source of infection to the community at large.

Where the naso-pharynx is affected, either primarily or secondarily through the nares or the pharynx, the constitutional symptoms are, as a rule, marked. This is in all probability accounted for by the great mass of absorbents in the naso-pharynx, where absorption takes place so easily that general septic poisoning quickly follows. Where the naso-pharynx is attacked by diphtheria, we usually meet with the most fatal results.

**The Larynx.**—In some cases the Klebs-Loeffler bacillus produces its effects first on the mucous membrane of the larynx. In these cases the mucous membrane of the nose and pharynx may never show any evidence of a pseudo-membrane. The first symptom, as a rule, is a cough of a harsh, ringing nature. The temperature may or may not be raised. As the toxic absorption is slight, on account of the locality affected, the constitutional symptoms are correspondingly mild. The child's symptoms are those resulting from laryngeal obstruction. There is dyspnoea, with retraction of the intercostal and supraclavicular spaces, and later of the epigastrium and the lower chest. This is accompanied by an increasing cyanosis. The child is very restless, is forced to sit up in order to breathe, and, for the same reason, bends forward with its head back. In these extreme cases, unless relief is speedily afforded, the child soon dies of suffocation. In another set of cases a slower form of suffocation may result from the extension of the membrane downward to the bronchi, while in still another set death may result from a complicating broncho-pneumonia.

A very prominent symptom in all forms of diphtheria may be cardiac weakness. In some cases the child may die suddenly without having presented any previous symptoms, or death may have been preceded by attacks of semi-collapse. In other cases there may be a weak, fluttering, intermittent pulse throughout the disease, which persists during convalescence. Under these circumstances the child should always be considered to be in a critical condition, as death, sometimes sudden, is liable to occur.

**COMPLICATIONS AND SEQUELÆ.**—There are a number of complications which arise in diphtheria besides those of laryngeal stenosis and cardiac weakness. The most serious of these are broncho-pneumonia and acute nephritis.

The form of pneumonia which complicates diphtheria is broncho-pneumonia, which, I have already told you, is produced, not by the Klebs-Loeffler bacillus, but by pyogenic cocci which have been inspired. Broncho-pneumonia is most frequent and most fatal in laryngeal cases which have been operated upon.

Albuminuria is so commonly met with in both the mild and the severe cases of diphtheria that it should be considered as a part of the disease rather than as a complication; as a rule, the greater the amount of albuminuria the more severe the case. Where acute nephritis complicates diphtheria it is not usually accompanied by œdema or anasarca.

Dysphagia may from the very beginning of the disease produce a profound impression upon the general nutrition. Otitis media occurs frequently.



Among the more common sequelæ are anæmia and chronic catarrh.

The most common and serious sequela of diphtheria is a peripheral neuritis, with its accompanying paralysis. This paralysis often does not appear until convalescence has been established,—perhaps in the third or fourth week from the time of the beginning of the attack. The paralysis may sometimes be merely of the muscles of the soft palate, in which case the fluids taken by the mouth are regurgitated through the nose; or it may have a general distribution, such as is seen in multiple neuritis. In the more severe cases of paralysis arising from this multiple neuritis, the lower extremities are affected and the knee-jerks are absent. The electrical reactions where the limbs are involved are the same as in peripheral neuritis from other causes.

The prognosis in these cases of post-diphtheritic paralysis is good.

DIAGNOSIS.—Recognizing that the same pseudo-membranous condition in the throat may occasionally be produced by the pyogenic cocci, as well as by the Klebs-Loeffler bacillus, the clinical diagnosis of a typical case of diphtheria is not difficult. A provisional diagnosis of diphtheria should be based upon the appearance in the throat of a pseudo-membrane, which usually appears first on the tonsils and has a tendency to spread to the uvula, soft palate, and pharynx. When in addition to this a nasal discharge is present and the glands of the neck are much enlarged, you have a picture which is not shown by any other disease. The most common difficulty met with clinically is in distinguishing between cases of acute follicular tonsillitis and diphtheria.

As I have already stated, the local lesions produced by the Klebs-Loeffler bacillus may be merely a catarrhal inflammation or those of a follicular tonsillitis. All such conditions, therefore, should be looked upon with suspicion until the absence of the Klebs-Loeffler bacillus has been demonstrated bacteriologically. Although a membranous laryngitis may be due to other causes than the Klebs-Loeffler bacillus, yet this is so rare that every case of primary membranous laryngitis should be considered to be diphtheria until it has been proved that it is not. A decisive diagnosis of diphtheria in any case can, therefore, be made only by determining the presence of the Klebs-Loeffler bacillus.

PROGNOSIS.—Diphtheria is an extremely fatal disease, especially in the septic and obstructive cases. The mortality varies decidedly in different epidemics and according to the age. Children under two years of age rarely recover. The rate of mortality seems to have lessened in cases where the antitoxin treatment has been thoroughly used. The symptoms which make the prognosis especially unfavorable are the extension of the membrane to the naso-pharynx or the larynx, extensive glandular enlargement, hemorrhage from the nose or into the skin, a high grade of albuminuria, broncho-pneumonia, and a weak heart. Morse, in an extensive study of the leucocytosis of diphtheria, has shown that it is of no prognostic value. The cases of neuritis invariably recover. The prognosis in all cases is



uncertain, and should be given with caution, as death from heart-failure is liable to occur at any stage of the disease.

A child who has had diphtheria is liable to suffer from the deleterious effects for months or even years.

PROPHYLAXIS.—All patients with diphtheria should be isolated until the Klebs-Loeffler bacillus has disappeared from the nose and throat. The time when this occurs varies from a few days to a number of weeks.

In order further to protect the community, all cases of sore throat should be examined, and if the Klebs-Loeffler bacillus is found the individual should be isolated. It is especially necessary to carry out this precaution in schools, where the conditions are so favorable for the spread of the disease.

The throats and noses of all persons exposed to diphtheria or caring for diphtheritic patients should be repeatedly examined for the Klebs-Loeffler bacillus, and if this is found they should be given immunizing doses of antitoxin, the amount and frequency of the doses to be modified as our knowledge increases. If in the future it is proved that the antitoxin may produce serious effects in certain individuals, these views must be modified to correspond to this additional knowledge. If the Klebs-Loeffler bacillus is found in these individuals, they should be isolated so long as the bacillus is present. To shorten the period of isolation, mild antiseptic gargles or douches should be employed. Whether the isolation of healthy persons who have the Klebs-Loeffler bacillus in their throat or nose is advisable or not is still a mooted question. Much confusion has arisen because of the so-called pseudo-diphtheritic bacillus. The weight of evidence at present, however, goes to show that it does not exist, and that the bacteria described are merely Klebs-Loeffler bacilli of diminished virulence. At any rate, even if the pseudo-diphtheritic bacillus exists, it is so rare that it may be safely excluded in clinical work. The fact that the Klebs-Loeffler bacilli found in healthy throats may not be virulent is not an argument against isolation, because it is well known that a non-virulent form may become virulent when transferred to a different soil. Examinations of many healthy throats have shown that the Klebs-Loeffler bacillus is a very rare inhabitant of the normal throat, and that when it is present diphtheria often develops later. Theoretically, therefore, although it may be impossible or inadvisable practically, it would seem wise to consider the Klebs-Loeffler bacillus virulent until it has been proved to be non-virulent, and to consider its presence a source of danger to the community until it is proved not to be.

In addition to what I have already said, I must impress upon you the importance of keeping the teeth in good order as a prophylactic measure, as well as keeping the mucous membrane of the nose and throat in a normal condition.

TREATMENT.—The treatment of diphtheria consists (1) in attending to the hygienic conditions; (2) in the administration of remedies, either by the skin or by the mouth, to combat the toxine which produces the constitutional symptoms; (3) in local applications to the nose, throat, or larynx, and in

measures directed to the general condition; (4) in operative measures to relieve obstruction in the larynx.

One of the most important parts of the treatment of diphtheria is the management of the room in which the patient is kept during the progress of the disease. It is well known that pathogenic organisms, such as the Klebs-Loeffler bacillus, do not thrive where they are exposed to sunlight and fresh air. The room should be thoroughly ventilated, and fresh pure air should be allowed to come continuously into it. It should also be one which has a sunny exposure.

In any treatment directed to the cure of diphtheria in young children we must remember that the disease is so exhausting that the treatment, as a rule, should be forced upon the child as little as possible. Any physical exhaustion produced by the treatment is to be considered serious in young children.

It is necessary perhaps to call your attention to the fact that much care should be taken both by the physician and by the nurse not to become infected themselves by the secretions from the mouth and nose of the patient. These secretions are especially dangerous if they happen to get into the eyes. It is probable that with extreme care there is not much danger of the spread of diphtheria in a household, as we know its tendency is not to disseminate itself in the surrounding atmosphere. Hence it is likely that with proper precautions it can be limited to the room in which the child is sick, and that if it extends beyond this room it has been carried directly by the hands or clothing of the nurse or the physician.

According to the knowledge of the present time, the most promising of all these forms of treatment is the second. This treatment is essentially comprised under what is called serum therapeutics. By serum therapeutics is meant the treatment of disease by injecting into the patient the serum of an animal which has been rendered immune to the especial disease, which is being treated, by means of inoculation with the toxine of that disease. The serum taken from the animals which have been rendered immune against diphtheria is called antitoxin serum. The serum is injected under the skin, usually in the thigh, and the place selected should always be one on which pressure is not exerted when lying in bed. The dose should be from 500 to 1000 antitoxin units, or 10 to 20 c.c. ( $\frac{1}{3}$  to  $\frac{2}{3}$  ounce) of the 1 to 50,000 serum, according to the age of the child and the severity of the disease.

The beneficial results of antitoxin are decidedly greater if the injection is made in an early stage of the disease than if made in the later, although even when administered late in the disease it sometimes produces wonderfully curative effects. When given early, within the first forty-eight hours of the disease, even where the membrane is spreading rapidly and inflammation of the glands with general systemic poisoning has taken place, one injection will often arrest the disease. Where improvement does not take place within twenty-four hours, a second dose, and, if necessary, a larger one, should be used. The sign by which we know that the antitoxin serum



is beneficial is the improvement in the general condition of the patient. The effect of the antitoxin on the pseudo-membrane is characteristic. The pseudo-membrane ceases to spread, frequently whitens, shrinks, shows a line of demarcation, and usually within the next three or four days becomes detached from the mucous membrane. The temperature usually rises after the injection, but in a few days falls to the normal by lysis. In the more severe cases a single injection of the serum does not work so quickly. In these cases the temperature falls usually by lysis after the second or third dose. The pulse becomes normal two or three days after the temperature has fallen. The irregularities of the pulse are not so frequent in diphtheria since the antitoxin treatment has been employed. The effect of antitoxin on the albuminuria is still *sub judice*, but it probably does not increase the likelihood of its occurrence. When there is a concurrent infection the antitoxin serum is less effective, since it does not counteract the toxic absorption due to other bacteria than the Klebs-Loeffler bacillus. It is not safe to assume, however, that there is a concurrent infection because other bacteria are found in the throat.

When the larynx is involved, with accompanying stenosis, the antitoxin serum is found to be very valuable, and has reduced the number of operative cases.

In connection with the antitoxin treatment no specific drugs given internally by the mouth are indicated. Stimulants should always be given freely in diphtheria. Of course, symptomatic treatment of any kind is not contraindicated.

The antitoxin has been found to have but little effect on the length of time during which the bacteria remain in the throat after the disappearance of the membrane.

Too few cases have as yet been observed to estimate the relative frequency of the occurrence of neuritis since the treatment by antitoxin has been introduced. Various skin and joint complications, accompanied by fever, occur in a certain proportion of the cases in which antitoxin is used. Albuminuria has been attributed to its use; but, as already stated, this question must still be considered as *sub judice*. Antitoxin is also said to cause serious and even fatal results in some cases. How much danger there may be in its use cannot as yet be estimated, but must be left to the future to decide. Careful clinical observations and autopsies on fatal cases in which it has been used can alone enlighten us.

The local treatment of diphtheria consists in thoroughly cleansing the mouth and nose with warm, non-irritating solutions, such as normal salt solution, or boracic acid four per cent. All strong and irritating applications to the throat in diphtheria are harmful.

The technique of the local applications to the throat and nose is important. The most simple, efficacious, and safe, and that which produces the least discomfort, is by irrigation. The same method—namely, by means of a fountain syringe—should be employed for either the throat or the nose,



except that in the former a larger hard-rubber nozzle should be used than for the nose, and one which is sufficiently long to pass over the base of the tongue.

Here is an illustration (Fig. 105) of the method of irrigation as employed in the Boston City Hospital and at the Willard-Parker Hospital in New York.

FIG. 105.



Irrigation of nose in diphtheria.

The child should lie on its side, and the water should be made to pass up one nostril and down the other until the stream runs clear. In some cases the child prefers to sit up while the irrigation is done. Ordinarily, the irrigation should be used once in two or three hours, perhaps with longer intervals at night. If the child resists this treatment, it may be advisable, in order to save its strength, to omit it for a time. This rule applies to all forms of local treatment.

Considerable suffering is at times occasioned by the enlargement of the cervical glands. Some patients prefer the application of ice poultices, others of hot flaxseed poultices. Either may be used if they produce the desired effect of reducing the discomfort.

Nutritive enemata made of peptonized milk, with stimulants, may, when retained, be an important adjunct to the treatment. Enemata, however, are often not retained. Digitalis may be used in cases where heart-failure is anticipated. In cases where there is a faucial paralysis the child may often, with success, be fed through the nostril by means of a soft-rubber catheter passed into the œsophagus; this method may also be used after intubation where there is unusual difficulty in swallowing.

Where measures are found to be necessary to reduce obstruction in cases of stenosis of the larynx, the child should be placed in an atmosphere of steam, and if this does not relieve the stenosis the sublimation of calomel

should be employed. In either case, however, we must remember that the child should not be kept in this atmosphere continuously, and should be watched carefully to see if it is speedily relieved of the stenosis; for if it is not, the continuous inhalation of steam in the comparatively small area of breathing space which exists in the tent that is used for this purpose may of itself be detrimental to the child's recovery, from lack of sufficient oxygen. When tracheotomy has been performed an atmosphere of steam is especially valuable.

The tent, as described by Dr. Northrup, who has used it so extensively in the Willard-Parker Hospital in New York, contains about fifty cubic feet of air. To extemporize a tent, a sheet is thrown over supports above the crib and allowed to fall over the four sides of the crib. The main point is to have a fairly large and tight enclosure. The apparatus for furnishing the steam or sublimation must be free from the danger of upsetting and of setting the tent on fire. For sublimation, a deep vessel, such as a wash-bowl, should have an alcohol lamp placed in it, and over its top a tin strip. Over the space where the flame of the alcohol lamp touches the under side of the strip a little, compact pile of calomel, sufficient for a single sublimation, is placed. Eight or ten minutes are required to volatilize the calomel, and the tent should be kept closed about fifteen minutes. A safe and satisfactory method is to volatilize in an ordinary crib-tent 0.3 gramme (5 grains) of calomel every two hours for two days and nights, and then prolong the intervals to three hours on the third day, four hours on the fourth day, and later three times a day, according to indications (O'Dwyer). It has been Dr. Morse's experience that 0.6 gramme (10 grains) every half-hour for four or five times will sometimes produce good results where the smaller doses have failed.

The nurse may easily become salivated from inhaling the calomel fumes, and should be cautioned in regard to this. The child should not be exposed to a sudden change of temperature when the sublimation is over. The room should be thoroughly aired after opening the tent, and it is well to remove the child to another room while this is being done. Young children do not, as a rule, suffer from ptyalism following this sublimation. Older children, after a number of days' treatment, may show a mild stomatitis, and sometimes diarrhoea. Chemically pure calomel is essential, as the impure drug may cause conjunctival irritation. If the fumigation has to be very prolonged, anæmia may be caused. This should be combated by iron, and if there is prostration, a little whiskey should be given before the sublimation.

The sublimation of calomel is indicated where the symptoms of laryngeal obstruction are urgent, and may be used alone or in conjunction with steam.

Where the antitoxin does not relieve the symptoms of stenosis, and where the progressive dyspnoea is not quickly controlled by steam or calomel sublimation, it is well not to delay operative interference. The operative means of relieving stenosis of the larynx is by intubation or by tracheotomy. The indications in either case are, according to Northrup, a progressive,



unremitting dyspnœa, when the labored breathing begins to produce sensible exhaustion, and when the supraclavicular and lateral thoracic retraction is marked. It would not be within my province to speak of the relative advantages of intubation and tracheotomy. Each operation has its strong exponents, and so much has been said in favor of both operations that the question as to which is best must be decided by the individual surgeon in the especial case. It is probable that the antitoxin treatment will increase the field for intubation in operative cases.

In the treatment of post-diphtheritic paralysis strychnine is the most valuable drug. Electricity, especially faradism, is also indicated.

The subsequent anæmia, which I have already referred to, should be treated in the usual way.

## DIVISION XIII.

### DISEASES OF THE ŒSOPHAGUS, STOMACH, AND INTESTINE.

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#### LECTURE XLIII.

##### INTRODUCTION.

BEFORE speaking in detail of the diseases of the stomach and intestine, a few general remarks are necessary to explain how limited is our knowledge of these diseases. Those diseases, however, which affect the œsophagus can easily be classified on a pathological basis, and are so few in number that they can be included in these general remarks.

**ŒSOPHAGUS.**—The diseases of the œsophagus are rare in infancy and early childhood. There may be congenital malformations, such as narrowing or dilatation. The swallowing of hot or corrosive liquids may cause obstruction, which is occasioned by a cicatricial stricture. Œsophageal stricture may also occur as a result of congenital syphilis. Pressure outside of the œsophagus may cause obstruction. These strictures, especially those of cicatricial origin, are accompanied by a great deal of muscular spasm, which at times is constant, and again relaxes. Thus, the child will swallow with comparative freedom at intervals, while at other times the obstruction appears to be complete. In addition to the inability to swallow, and the consequent regurgitation of the food, the secretion of saliva and mucus is often very profuse, and causes symptoms of distress and choking.

The diagnosis and treatment of these cases are effected chiefly by means of bougies; but, as much harm may come from these instruments, and as especial surgical knowledge is required to use them and to decide whether œsophagotomy should be performed, I shall not dwell on this class of cases.

An inflammatory condition of the œsophagus is said to occur in young infants, and is spoken of as *œsophagitis*. It is rare. The symptoms, as described by Billard, are unwillingness to nurse, crying, immediate regurgitation after beginning to suck, and often some tenderness about the neck on pressure. The prognosis is bad.

It is quite common for children to swallow various *foreign bodies*, such



as buttons and pins. These bodies may either be caught in the back of the throat or lodged in the œsophagus, instead of passing through to the stomach. A careful examination of the throat with the finger should first be made, and if the foreign body is not detected in the throat the œsophagus should be explored carefully with a bougie, and the foreign body is then usually pushed through into the stomach, unless it is thought wiser to remove it with the bristle probang. The diet for the following twenty-four to forty-eight hours, or until the body has been passed through the intestine, should be such as will give sufficient consistency to the fæces to protect the intestine from injury while the body is being passed over its surface. Various

FIG. 106.



Congenital dilatation of œsophagus, female, 10 weeks old ( $\frac{1}{3}$  natural size).

preparations of the cereals are useful for this purpose. If necessary, a dose of oil can be given, but, as a rule, active treatment is contra-indicated.

I have here a specimen of the œsophagus and stomach of an infant (Case 406) ten weeks old which shows the condition of congenital dilatation of the œsophagus (Fig. 106).

The infant was healthy at birth, and its mother had a plentiful supply of breast-milk. During the first two or three weeks of its life nothing abnormal was noticed about it, except that it vomited occasionally. When it was four weeks old it began to regurgitate, vomited the milk frequently, and lost in weight. The faecal discharges showed that the milk which reached the stomach and intestine was fairly digested, but the discharges were infrequent and small in number. It was weaned when it was nine weeks old, and small amounts of milk, carefully modified in various ways, were given to it. No improvement in the symptoms followed this treatment, and although at times a small quantity of milk would be retained, yet, as a rule, after a few minutes the milk was regurgitated. The infant had no other symptoms, but rapidly lost in weight, and finally died of exhaustion.

The post-mortem examination was made by Dr. Whitney, and the only pathological conditions found were, as you see, in the œsophagus. The last two inches of the œsophagus were dilated into a more or less cylindrical swelling, with marked thinning of the walls and atrophy of the mucous coat. A dilatation had been formed in which evidence of a small area about to perforate into the mediastinum was found. The entire stomach, as well as its cardiac and pyloric orifices, was markedly contracted, apparently from lack of use.

**STOMACH AND INTESTINE.**—Our knowledge of the diseases of the stomach and intestine is exceedingly limited, and is especially so where infants and young children are concerned. The classification of these diseases on a pathological basis has been proved to be inadequate, and in like manner a classification on the basis of symptoms is insufficient. Bacteriological investigations, however, have advanced our knowledge to such an extent that we may hope in the future to be able to classify these diseases on an etiological basis. The terms dyspepsia, dysentery, diphtheritic, croupous, and others have become almost unmeaning, and should be replaced by terms more closely connected with the etiology of the disease.

Accordingly, the American Pediatric Society requested Dr. Holt and myself to prepare a nomenclature which would correspond more nearly to our present knowledge of this exceedingly difficult subject. I wish especially to emphasize the value of Dr. Holt's work, which has aided me so much in my own studies on this subject. The classification finally adopted by the Society was one which especially relates to infants and young children, and you must remember that in what I am about to say concerning this important class of diseases I am dealing especially with this early period of life. The diseases of the gastro-enteric tract as they occur in older children resemble so closely those of adults that they need not occupy a prominent place in lectures on children, especially as the pathology and symptoms of this later period of life differ very materially from those of the earlier periods. These differences are still more strongly marked from the fact that children succumb much more readily to the early stages

of a disease than do adults, and may die before the later and more characteristic lesions and symptoms of the disease have developed. There are certain known facts resulting from the anatomical and physiological peculiarities existing in infancy which play a significant part in all these diseases. It is well, therefore, first to explain the general principles which influence the symptoms and prognosis of these diseases before attempting to describe each disease separately. In many cases we can arrive at only approximate conclusions as to the actual lesion which exists and the prognosis which should be given. A practical clinical diagnosis should be made according to the region where the stress of the lesion exists, rather than to the pathological lesions which are present.

**GENERAL ETIOLOGY.**—In the present state of our knowledge it is not practicable to discuss in detail the various supposed causes of gastro-enteric disturbances. We can suppose that these disturbances may be due to nervous conditions which may act alone or may render the tissues vulnerable to bacteria. Some of these diseases are caused by specific organisms, while others are due to a number of organisms. These bacteria act either of themselves or through their products.

In a general way, these diseases can be classified as functional and organic. The organic class may be divided into inflammatory and non-inflammatory diseases, although the boundary-line between these two conditions is at times very doubtful. A prominent and important peculiarity of these diseases as they occur in infancy is, as would naturally be expected at this early period of development, a variety of symptoms which are produced by reflex causes. By the term reflex we mean peripheral irritation with a resulting action. By functional we mean a disturbance of the function of the organ without a known lesion. By organic we mean a known lesion.

In addition to these cases are others which, as yet imperfectly understood, seem to be produced by certain morbid products eliminated from the blood by the gastro-enteric tract, as, for example, urea. This etiological factor can be spoken of under the term *eliminative*.

**GENERAL PATHOLOGY.**—The general pathological anatomy of the gastro-enteric tract of infancy and early childhood is essentially that of the ileum and colon. In those cases in which the more severe lesions are present the stress of the lesion is usually in the lower ileum and the colon, and very frequently in the colon only. For this reason the terms ileocolitis and colitis seem more descriptive than ileo-enteritis and enteritis. The pseudo-membrane in ileocolitis is often extensive, but sloughing and perforation are exceedingly rare in young children. It is at present believed that not all ulcers of the gastro-enteric tract are necessarily inflammatory. The great number of lymph-nodes and the abundance of the lymphatic plexuses are the principal anatomical conditions which influence the pathology of the enteric tract in early life.

**GENERAL BACTERIOLOGY.**—The knowledge of the different bacteria which occur in the gastro-enteric tract, and of the connection which they



have with the different diseases, is at present, with few exceptions, uncertain and unreliable. There is little doubt that the bacteria may find their way, by means of the stomach, to the intestine, and that the acid secretion of the stomach which they meet in their way through it is not sufficient to prevent their arriving alive in the intestine. We know that these bacteria play such an important rôle in their etiological relations to the various diseases that full weight must be given to their presence when we are treating the disease. It would seem that the bacteria which are commonly found in the intestine when it is in a normal condition do not cause any abnormal conditions; but when the intestine has become irritated, from mechanical or thermic causes, the bacteria are able to penetrate its mucous membrane, become noxious, and produce abnormal symptoms, often of a serious nature.

GENERAL SYMPTOMATOLOGY.—Vomiting as a symptom is often very misleading in early life, so far as the differential diagnosis between the stomach and the intestine is concerned, as it frequently occurs from disturbance in any part of the gastro-enteric tract, and should not be considered as indicative of any one disease. Serious symptoms during life are often proved at the autopsy to have been produced by no pathological lesion, while grave lesions may be found at the autopsy where the intestinal symptoms during life were very mild.

Marked diarrhœa may exist during life and no lesions be present at the autopsy. Serious lesions may exist, and yet no blood appear in the dejections. Blood may appear in the dejections, and yet no serious lesion exist, the hemorrhage being only temporary, and comparable to epistaxis.

GENERAL DIAGNOSIS.—The observation of the temperature is very important for the diagnosis of these diseases. As a rule, an elevated temperature of short duration points towards functional and toxic disturbances, while an elevated temperature long continued points towards inflammatory lesions.

Intestinal discharges are often very misleading for diagnosis.

Having considered and accepted these general principles relating to diseases of the gastro-enteric tract in infancy, the American Pediatric Society adopted the following classification (Table 109), as presented to them by their committee. This classification must be understood to be merely provisional, and is for the purpose of aiding those who are interested in this subject to work with uniformity.

At the same time it is believed that it is a great advance upon the unmeaning and misleading nomenclature now current.

On examining this table (Table 109) you will see that whenever the etiology has been definitely determined it is made to designate the disease, but the true etiology is still unknown in so many cases that other terms have of necessity been used, the names simply representing the extent of the knowledge we have of the especial disease.

The diseases of the gastro-enteric tract may, on this basis, be divided into diseases of the stomach (gastric), diseases of the intestine (enteric), and

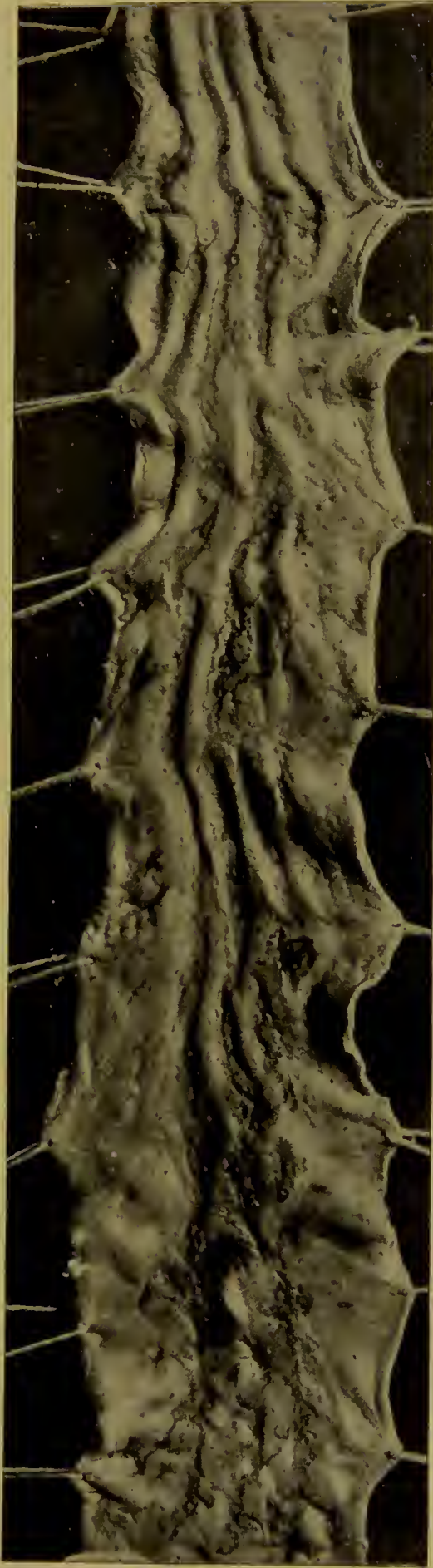
TABLE 109.

GASTRO-ENTERIC TRACT.		CENTRAL.		IDIOREFLEX.	
GASTRIC . . . . .	Developmental . . . . .	{ Malformations, Malpositions.		{ From many causes, as tubercular meningitis, heat, cold, fright, etc.	
	Functional . . . . .	{ Acute . . . . . { Nervous (vomiting) . . . . . { Indigestion.		{ Irritation from foreign bodies, food, or otherwise.	
	Organic . . . . .	{ Chronic . . . . . { Indigestion.		{ Pseudo-Membranosa.	
		{ Eliminated.		{ Such as, according to Wollstein in Archives of Pediatrics for July, 1892, is met with in exhausting diseases, atelectasis, diphtheria, tuberculosis, etc.	
ENTERIC . . . . .	Developmental . . . . .	{ Malformations, Malpositions.		{ Catarrhalis.	
	Functional . . . . .	{ Acute . . . . . { Nervous (exaggerated peristalsis causing diarrhoea).		{ Corrosiva.	
	Organic . . . . .	{ Chronic . . . . . { Indigestion . . . . . { Incontinence.		{ Pseudo-Membranosa.	
		{ Eliminated.		{ Such as, according to Wollstein in Archives of Pediatrics for July, 1892, is met with in exhausting diseases, atelectasis, diphtheria, tuberculosis, etc.	
GASTRO-ENTERIC TRACT.	Developmental . . . . .	{ Malformations, Malpositions.		{ Catarrhalis.	
	Functional . . . . .	{ Acute . . . . . { Nervous (exaggerated peristalsis causing diarrhoea).		{ Corrosiva.	
	Organic . . . . .	{ Chronic . . . . . { Indigestion . . . . . { Incontinence.		{ Pseudo-Membranosa.	
		{ Eliminated.		{ Such as, according to Wollstein in Archives of Pediatrics for July, 1892, is met with in exhausting diseases, atelectasis, diphtheria, tuberculosis, etc.	





FIG. 107.



Colon showing presence of bismuth which had been given by the mouth.  
(Page 839.)



the disturbances which arise from animal parasites. The diseases are then divided into those which arise from developmental, those which arise from functional, and those which arise from organic causes. The organic diseases are subdivided into non-inflammatory and inflammatory, and the functional and organic diseases into acute and chronic.

GENERAL TREATMENT.—In the treatment of these diseases we should endeavor to carry out four general rules: (1) to combat the serious conditions already referred to; (2) to dislodge the bacteria as quickly as possible, perhaps by laxatives and irrigation; (3) not to introduce into the gastro-enteric tract for a certain period food which may prove a favorable culture ground for the bacteria, since it has been shown that where the food is sterile when it enters the gastro-enteric tract it is quite effective in reducing the number of bacteria in the intestine; (4) to introduce such drugs into the gastro-enteric tract as may, by their anti-fermentative and germicidal powers, diminish the action of or destroy the bacteria. This last rule is, however, very difficult to carry out, and, with our present knowledge of drugs and their administration, practically impossible. It is true that we know that subnitrate of bismuth is an anti-ferment, and that it reaches the part of the enteric tract which we know to be most affected in enteric disturbances characterized by fermentation. In proof of this I need simply refer you to this intestine (Fig. 107, facing page 838) of an infant, given to me by Dr. Holt, to illustrate this point, where bismuth had been given, and where at the autopsy the bismuth was found thickly coating the mucous membrane of the small intestine, and also appearing in the large intestine. It is, however, questionable whether in any case the attempt to kill the bacteria by the internal administration of drugs has been successful. Preparations, such as salol, which are known to be broken up into their carbolic acid components on reaching the intestine, cannot with safety be given to the infant in doses large enough to kill the bacteria, for in such doses there may be serious results from poisoning. We can, however, possibly, by means of these germicidal drugs, produce a condition in the intestine which, though not conducive to the death of the bacteria, may yet be so unfavorable for their growth as to aid our treatment when we are endeavoring to dislodge them. Nothing definite has, however, as yet resulted from using drugs for this purpose, and, so far as I can judge, the danger of treating infants or young children in this way is greater than the good that may result from it.



## LECTURE XLIV.

## DISEASES OF THE STOMACH.

FROM what I told you in the last lecture you will understand how difficult it is to make a differential diagnosis between gastric disease and gastro-enteric disturbance. \* The only symptom which definitely shows the stomach to be involved, whether from reflex, functional, or organic conditions, is vomiting, and, as we know that in many cases vomiting is caused primarily by disturbance of the intestine, we really have no symptom which represents gastric disease alone. The difficulty of locating disease in the stomach is rendered still greater by the fact that serious organic lesions may exist in the stomach without any symptoms whatever, whether of vomiting, pain, or tenderness. We must, therefore, be exceedingly cautious in making a diagnosis of diseases of the stomach.

Diseases of the stomach may arise from *developmental*, *functional*, or *organic* causes.

**DEVELOPMENTAL.**—Under developmental affections of the stomach are included *malformations* and *malpositions*. A malformation of the stomach may be represented by a narrowing of either the cardiac or the pyloric orifice, or by constrictions in various parts of the ventral cavity, which are known as hour-glass contractions. A malposition of the stomach may be met with in various places, one of which is in the thoracic cavity. These malpositions, however, are exceedingly rare, and of pathological rather than of clinical interest, as the diagnosis can scarcely be made during life.

**FUNCTIONAL.**—The functional diseases of the stomach play a great rôle in infants and in young children. They may be of an *acute* or a *chronic* variety, or may be what I have spoken of as *eliminative*. The latter class, in which certain morbid and irritating substances are supposed to enter the stomach as though it were an excretory organ, may in the future explain many of the rather obscure gastric symptoms which arise in early life, but at present our knowledge concerning this class of cases is so slight and indefinite that they need merely be alluded to. Acute functional gastric symptoms may be produced by a number of causes, but in general they are to be understood as arising from a *nervous* condition represented by vomiting or by a disturbance of the function of digestion, which had best be spoken of, until more is known of the subject, as *simple indigestion*.

**NERVOUS (Vomiting).**—Vomiting may arise from gastric or from intestinal irritation in many diseases, such as tubercular meningitis, from heat, cold, fright, and from other causes. Direct irritation, from foreign bodies, food, or otherwise, may produce a reflex form of vomiting. In these cases the cause, if possible, should be removed, and the stomach given

a complete rest until the nervous disturbance has subsided. As a rule, no internal remedies are indicated in these cases, except an emetic where the vomiting arises from the reflex causes just described, or, if necessary, lavage.

There is one form of vomiting, however, which is of such importance that it must be spoken of as a disease by itself. There is no name which can be given to it except that of *persistent vomiting*, as no single definite cause nor any pathological lesion has ever been proved to produce it. It has not even been shown that it is a primary disturbance of the stomach. In fact, in many cases it is possible that the source of irritation is entirely outside of the stomach, and perhaps connected with the great sympathetic ganglia, such as the solar plexus.

ETIOLOGY.—The inciting cause of the vomiting in most cases is obscure, but is evidently very varied. It does not seem to be produced especially by errors of diet, but, on the contrary, occurs in children whose diet has been most carefully regulated. Undue exposure to cold, fright, and excitement all appear to me to have sometimes an etiological influence on the disease. This form of vomiting may occur at any age. I have met with cases in young infants and throughout the whole period of childhood. The attacks may occur not only in delicate, nervous children, but also in those who are quite vigorous.

SYMPTOMS.—The attack is very apt to come on suddenly, the child being previously in good health and not having shown any digestive disturbance. The period over which the disease extends and the intervals of the vomiting during the attack vary considerably, but in those cases which have come under my notice they are somewhat as follows. The child, without any especial warning, begins to vomit, and at first the vomitus will simply be the remains of food which still happen to be in the stomach. It will continue to vomit quite regularly every fifteen or thirty minutes. This may last for ten or twelve hours; the intervals then grow longer, and sometimes the vomiting will cease for twelve or fifteen hours and then begin again. Occasionally a little mucus appears in the vomitus, but not to any great extent. As the disease progresses, a slight amount of bile usually appears in the vomitus. A very prominent symptom is thirst, the child crying continually for water, and vomiting it soon after it is taken. As a rule, the temperature in these cases is normal or subnormal. The pulse varies, but is very apt to be slow, sometimes intermittent, and may become weak. After the first twenty-four hours the child emaciates rapidly, looks very ill, and becomes apathetic.

Unless the disease is unwisely treated by endeavoring to introduce food or drugs into the stomach, it will usually prove to be self-limited, and will run its course in two or three days. In some cases the length of the attack is much shorter, being comprised within twenty-four hours, while in others it may last for many days. The recovery is often as sudden as was the onset of the disease. As soon as the vomiting has stopped, the appetite



returns; there are no special symptoms of indigestion; the child takes its food well, and the emaciation disappears rapidly. Relapses occasionally take place.

DIAGNOSIS.—The diagnosis of *persistent vomiting* is often difficult, more on account of a lack of sufficient knowledge concerning the disease than from much evidence of the existence of the diseases which it is supposed to simulate. In these cases an examination of the abdomen should be made at once, including a rectal examination. This is necessary in order to exclude such sources of vomiting as intussusception and appendicitis. The absence of any marked increase in the temperature and a careful examination of the thorax will in most cases exclude the sudden onset of some pulmonary disease or of the acute infectious diseases. The disease which is most commonly suspected in these cases is tubercular meningitis. In some instances, after the disease has lasted for two or three days, the resemblance to tubercular meningitis may be quite striking; but if the whole course of the affection is taken into consideration, the diagnosis soon becomes clear. In persistent vomiting the face and general appearance of the child indicate nausea rather than the apathy which would be present in tubercular meningitis. The mind also, in contradistinction to what takes place in the latter disease, is clear, the child remaining quiet merely because it is exhausted. The great thirst which I have already mentioned as being present in persistent vomiting also aids materially in the differential diagnosis from tubercular meningitis. The sudden onset of the vomiting in a previously healthy child is quite different from the slow progress and the occasional vomiting of a cerebral type met with in tubercular meningitis.

After the first twenty-four hours, persistent vomiting is readily differentiated from attacks of simple indigestion, as where the vomiting arises from indigestion the stomach is speedily relieved, and the vomiting does not continually recur without apparent cause, as is the case where persistent vomiting is present.

Persistent vomiting is also very commonly diagnosticated as acute gastric catarrh, but in the latter disease the heightened temperature, coated tongue, pain, and tenderness in the epigastrium will, after the first twenty-four hours, allow us to differentiate the two diseases.

PROGNOSIS.—The prognosis of persistent vomiting varies according to the age of the individual affected. In young infants, especially in those whose vitality is weak, it may prove to be a very serious disease, from the exhaustion which invariably arises in the first twenty-four hours. The rule is that the younger the individual the more prostrating and serious is the disease. Even older children are at times so prostrated by the continuous vomiting that grave doubts are often entertained as to their ultimate recovery. In general, however, the prognosis in these cases is good, and, although I have met with a number of them, I have never seen the disease result in death.

TREATMENT.—The treatment of persistent vomiting is essentially star-



vation during the first twenty-four hours. The child should be kept perfectly quiet in a darkened room. If after twenty-four hours the vomiting still continues, or even before if there appears to be much exhaustion, or if the child is restless and sleepless and has an intermittent pulse, hydrate of chloral and bromide of potassium, dissolved in brandy and water, should be given by the rectum. These are intended to procure sleep and to stimulate the nervous centres. As a rule, however, the child is quiet, and sleeps in the intervals of the vomiting, and, as the disease usually attacks an infant or a child who has been perfectly well, cardiac weakness is not commonly shown in the first forty-eight hours. No food and no drugs should be given by the mouth. After forty-eight hours, small enemata of peptonized milk can be given, and when the disease appears to have run its course, as it often does in three or four days, small quantities of a carefully modified alkaline milk can be tried cautiously by the mouth. A mistake is usually made in the treatment of the disease in feeding by the mouth too early.

I shall speak of a few illustrative cases of this disease which have come under my notice, as a knowledge of them will be of great use to you in your practice.

The first (Case 407) was an infant, eight months old, strong and healthy, whose food had always been the milk of a wet-nurse. Without any previous symptoms the infant began to vomit, and continued to vomit every fifteen minutes for twelve hours. The intervals then became longer, and the vomiting ceased entirely on the third day of the attack. During the attack the infant emaciated rapidly, so that it looked as though it were in the last stages of some wasting disease. It lay perfectly quiet and slept in the intervals of the vomiting. Its mind was clear. Its temperature was subnormal, and its pulse weak and intermitting. It was treated by rectal enemata of brandy, peptonized milk, and bromide of potassium.

The infant had several attacks of this kind in each of the following years of its life until it was five or six years old, when it would sometimes go for six months or a year without an attack. As it grew older the attacks became less severe, and when it was ten years old they ceased entirely.

The next case (Case 408) was that of a girl, twenty-two months old, whom I saw in consultation with Dr. Joseph Stedman. She was perfectly well before the vomiting began. Her temperature was normal: the pulse was slightly accelerated at first, and later became slow and intermittent. During the first four days of the attack the vomiting was almost continuous, and she became so weak and exhausted on the fourth day that it was feared she might die suddenly. There were great restlessness, dilated pupils, throwing of the head backward, slow pulse, and normal respirations. The emaciation was rapid. The urine was scanty. On the fifth day, the vomiting having continued, she fell into a state of collapse, the pulse was hardly perceptible, her countenance was ghastly, and her extremities were cold. At one time after a severe attack of vomiting she became cyanotic, and was almost stifled by tenacious mucus. This, when vomited, appeared to invade the larynx, so that it seemed as though her life was saved a number of times by the prompt action of an experienced nurse. On the sixth day the vomiting grew less, and on the seventh day it ceased. She was not, however, able to be up and about until the eleventh day, and was not entirely well until the third week from the time that she was attacked. The treatment in this case was the same as in the previous one.

A third case, a boy (Case 409), nine years old, was seen by me in consultation with Dr. F. B. Harrington. This boy was attacked suddenly with vomiting as described in the

previous cases. The duration of the attack was about two weeks. The prostration was extreme, and the boy's strength was supported solely by enemata, as at no time during the two weeks could anything be retained by the stomach.

These last two cases were unusually protracted.

**ACUTE GASTRIC INDIGESTION** (*Acute Dyspepsia*).—By indigestion we mean a disturbance of the gastric secretions interfering with the function of the stomach to such a degree as to cause morbid symptoms. Exactly what this disturbance is in infants and young children has not yet been clearly proved. The cause of acute indigestion in infants, and in almost every case in young children, is the food which is given to them. This is especially noticeable in the first year. The ages at which indigestion most frequently occurs in this period are, first, in the early days of life, when the equilibrium of the breast-milk has not been established; second, in the middle of the first year, when the breast-milk is so apt to be replaced or supplemented by some other food; and, third, at the end of the year, when entirely new articles of diet are usually given to the infant.

**SYMPTOMS.**—The symptoms of acute indigestion are extreme pallor, nausea, eructations of gas, a general appearance of discomfort, due probably to the pain induced by the development of gas in the stomach, with its resulting distention, and finally vomiting. If the diet is exclusively of milk, the vomitus will usually contain large curds of the coagulated proteids. In connection with the gastric disturbance there is commonly constipation, although sometimes there may be a relaxed condition of the bowels. The faecal discharges accompanying these attacks are of an abnormal color, usually a mixture of green, white, and yellow, and of sour odor. There is little or no fever. At times the symptoms are so severe that the infant looks as though it were going to die. In rare cases also reflex symptoms of a serious aspect may arise, such as I have already described when speaking of *asthma dyspepticum* (page 750).

**DIAGNOSIS.**—Sometimes the diagnosis is obscured by the absence of vomiting, but the pallor and nausea are usually of sufficient prominence to allow us to decide that the seat of the disturbance is the stomach. An emetic, such as one-half to one teaspoonful of wine of ipecac, usually relieves the symptoms promptly and makes the diagnosis clear.

**TREATMENT.**—The treatment of acute indigestion is to empty the stomach, to give a mild laxative in order to clear away the undigested food, and to regulate the diet. The laxative may be one or two teaspoonfuls of castor oil, an eighth to a tenth of a grain of calomel for four or five doses, or a teaspoonful of liquid magnesia. If the food has been breast-milk, an analysis of the milk should be made at once, and the proper modification of the milk, according to the rules which I have already given you, should be carried out. If the infant is being fed on an improperly modified milk, or if improper food of any kind has been given to it, a recurrence of the attacks can easily be obviated by a modification of the elements of the food which seem to have produced the disturbance.



Thus, in a number of cases I have found that whenever the infant's food was modified so as to raise the percentage of the sugar above 5, acute indigestion followed. In like manner in certain cases the percentage of the fat had to be reduced to 3, or perhaps 2.5, and the proteids even as low as 0.45, for a number of weeks until the digestive function of the stomach became normal.

In older children the symptoms are similar to those which I have just described, and the diagnosis and treatment the same as in the infant, for there is no way by which an attack of acute indigestion can be so surely prevented from recurring as by at once placing the child for several days on an exclusive diet of a milk modified in such a way as to contain a percentage of from 2 to 5 of fat, 5 to 6 of sugar, 1 to 2 of proteids, and 10 of lime water.

**CHRONIC GASTRIC INDIGESTION (Chronic Dyspepsia).**—If the attacks of acute indigestion are allowed to occur frequently from lack of proper treatment, a subacute or chronic form of the disease develops.

**SYMPTOMS.**—In infants the symptoms of chronic indigestion are much less severe than those of the acute form. The infant is apt to vomit after taking its food, to be restless, fretful, and either to lose in weight or not to gain. Its sleep will be very much disturbed, apparently by pain from flatus. In chronic indigestion the bowels are apt to be constipated, but this is not always the case. The chronic indigestion of older children presents a somewhat different aspect. The temperature is at times somewhat heightened. The tongue is apt to be coated, and the breath to have an odor. These children do not vomit so frequently as do infants. They lose in weight, become fretful, and get tired easily.

**TREATMENT.**—I have seldom found the use of any especial drug to be of much benefit in these cases of chronic indigestion. In quite a number of cases of both acute and chronic indigestion, before any food is introduced into the stomach it is often wise first to wash out the stomach thoroughly (lavage). This procedure is especially indicated if the indigestion has produced continuous vomiting.

The technique of washing out the stomach is very simple. A soft rubber catheter with a double eye, No. 21 French scale, as recommended by Dr. Holt for infants under six months, and No. 25 for older children, is attached by means of a piece of glass tubing 7.5 cm. (3 inches) long to another rubber tube which is 50.5 cm. (20 inches) long attached to a funnel, preferably of hard rubber, and capable of holding from 90 to 120 c.c. (3 or 4 ounces). The infant is seated upright in the nurse's lap, with its head inclined forward and resting on the nurse's arm. Its arms are controlled by a towel pinned around them. The catheter, having been wet with warm water, is easily passed over the base of the tongue into the stomach. As there is often considerable gas in the stomach, the funnel should be raised as high as possible above the infant's head, in order that the gas may pass out from the stomach. From 90 to 120 c.c. (3 or 4 ounces) of sterilized



water should be poured into the stomach by means of the funnel. The funnel is then depressed below the level of the stomach, and the gastric contents will in this way be siphoned out. As the curds are often too large to pass through the eye of the catheter, a number of washings will often be necessary to break them up. By washing out the stomach not only are the irritating substances which are producing the indigestion removed, and the mucous lining of the stomach left free to recover its normal condition, but it is also possible to have a chemical examination of the contents made. Clinically, however, the latter is not necessary, although it is of great interest physiologically. No food should be introduced into the stomach for at least two hours after the washing. The washing of the stomach is almost entirely free from danger, and, in addition to being an important part of the treatment of indigestion, is often of great use where poisonous substances have been swallowed.

This method of treating disturbances of the stomach is more valuable in young infants than in older children, because the latter resist so vigorously that the remedy is often of more harm than good. The tube can, however, usually, even in older children, be introduced by aid of the ordinary gag which is used for intubation. Two assistants are usually necessary in introducing the tube in older children, while in infants one assistant is sufficient. In some cases it is found necessary to introduce the tube through the nose. The tube should be passed into the throat rapidly, since the gagging and vomiting occur chiefly when the tube touches the pharynx. There is usually an escape of gas or gastric contents as soon as the tube enters the stomach.

When the inflow of water through the tube is shown to be too rapid, by the fact that the infant holds its breath too long, or by its crying, vomiting, or coughing continuously, the flow should be stopped for a short time. Care must also be taken not to introduce the catheter too far into the stomach, as it may bend on itself and interfere with the flow of the returning water and gastric contents. If the gastric contents are expelled along the side of the tube rather than through it, the tube should be withdrawn until the vomiting has ceased. There seems to be no danger of passing the tube into the larynx, or of perforating the stomach with it.

Lavage is contra-indicated where there is cardiac disease or any severe pulmonary disturbance, and when the introduction of the catheter continues to excite vomiting it should be used with extreme caution. The fact that the infant is in a feeble condition is not a contra-indication to this treatment.

In connection with lavage it is well to speak of forced feeding (gavage) in the treatment of infants and young children. In cases of acute and chronic indigestion, and also where a catarrhal condition of the stomach is present, the infants at times refuse to take any food whatever. This does not occur merely where the disturbance is in the stomach: I have frequently met with it in severe cases of all kinds of disease. In a

number of instances, where the infants would probably have died of starvation had not gavage been employed, this means of providing for their nourishment has been very successful. Forced feeding may sometimes have to be employed for a number of days, and even weeks, before the child will of itself swallow again.

The technique of gavage is similar to that of lavage. The same apparatus is employed, but the child should be placed flat on its back in bed, and its head held by an assistant. The catheter should be passed into the stomach rapidly, the funnel raised up in the air for a few minutes in order that the gas may escape, and the amount of food adapted to the age of the child should then be poured into the funnel. As the last of the food disappears from the funnel, the catheter is pinched tightly and quickly withdrawn. This precaution is important, in order that the pharynx shall not be irritated either by the slow withdrawal of the catheter or by the trickling of the remains of the fluid, as vomiting may in this way be excited.

One of the advantages which has resulted from the use of the stomach-tube is the knowledge we have acquired of the time which the food remains in the stomach at different ages. Thus, it has been found that during the early weeks of life the stomach is nearly emptied in an hour, while in older infants two hours are required for the same process. This knowledge is especially valuable when we are regulating the intervals of feeding in premature infants, and in infants during the first six months of life. These intervals I have already given in my lectures on Premature Infants and on Feeding.

Where other means can be employed, they are preferable to the stomach-tube. I have found in most instances where infants or children refuse to take their food that the simplest way of forcing it upon them is to pinion the arms with a towel and have the nurse hold the child half reclining in her lap. Sometimes an assistant is needed to hold the head, but this is often unnecessary. Simply pressing the child's nostrils with the thumb and finger will cause it to open its mouth, and the food can then be poured in with a spoon, or, as I have done in a number of cases, by means of a dropper with a large end. A child two and one-half years old, who has recently been under my care, for several weeks would not take any food without being forced to do so. Although this child was very ill with pneumonia, involving both lungs, it was fed every two or three hours, night and day, by this method. After the first two or three feedings it did not resist, and the nose did not have to be pinched, all that was necessary being to threaten to do so. 120 c.c. (4 ounces) of milk were, after a little practice, introduced by means of the dropper into the child's stomach in five or six minutes.

I have found that the most speedy cure of chronic indigestion is to give the child a carefully modified alkaline milk. In some cases it will be necessary to reduce the fat or sugar, in others the proteids, but in every case, as soon as it is determined which of these elements in full strength



does not suit the individual digestion, an improvement in the symptoms will soon follow the reduction of the percentage of that element. After the indigestion has been relieved by this means, other articles of diet adapted to the age of the child can gradually be added.

In addition to the direct treatment of the stomach, the intestinal disturbance which almost always accompanies the gastric indigestion should be relieved by occasionally giving a dose of some mild laxative, preferably one of the salts of magnesia. This latter treatment is indicated not only for children, but for young infants, because, when there is gastric indigestion, the undigested food which passes into the duodenum is a prolific source of intestinal disturbance. This, by adding to the discomfort of the child, weakens it, and tends to prolong the gastric indigestion.

**ORGANIC.**—The organic affections of the stomach may be divided into non-inflammatory and inflammatory. They are, in my experience, very rare in comparison with the functional diseases which I have just described.

**NON-INFLAMMATORY.**—The non-inflammatory conditions of the stomach comprise a diminution in the size of the organ, mechanical dilatation, ulcers, and new growths.

**Contraction of the Stomach.**—In certain cases the gastric capacity of the stomach is decidedly diminished. This diminution in the size, as a rule, depends upon a lack of use, such as occurs in infantile atrophy. Sufficient food to fill the stomach is not taken, and in this way the stomach is not called upon to perform its normal work. In cases, also, where there is continuous vomiting, this same lack of use may produce a diminution in the size of the stomach. These cases are of pathological rather than of clinical interest, as they can seldom be diagnosticated, and their treatment is essentially that of the special disease to which they are secondary.

**Dilatation of the Stomach.**—Dilatation of the stomach is rather more common in infancy than in older children. It may rarely arise from some malformation, such as a stenosis of the pylorus, but in most cases is the result of errors in feeding. It is more apt to occur where the infant is not nursed, unless especial care is taken to give the infant the quantity of food which is adapted to its age and gastric capacity. When the infant is nursed, the breast seems to provide the amount of food which is suitable. Dilatation from errors in feeding may be caused by the fact that the food is not adapted, either in quality or in quantity, to the age of the individual infant. Where the quality is at fault, the nutrition of the tissues of the stomach is interfered with, and its walls become weak, and are thus more easily distended by the gas which results from the abnormal changes in the food. In this way dilatation occurs. This class of cases is notably represented in the disease *rhachitis*, where dilatation of the stomach takes place very readily.

Where the quantity of the food is not properly adapted to the size of the stomach, dilatation can take place in even a healthy infant. I have already demonstrated to you, in my lectures on Development and Feeding,



the size of the stomach at different ages, and the amount of food which it normally holds. I shall, therefore, not repeat what I explained to you so fully at that time, but shall merely impress upon you the great importance of carefully regulating the amount of food which is given at each feeding during the first year of life.

**PATHOLOGY.**—The pathological condition which exists in cases of gastric dilatation is well represented in this stomach (Fig. 108).

FIG. 108.



Dilated stomach. Rhachitic infant, 7 months old. (Natural size.)

It was taken from an artificially fed rhachitic infant (Case 410), seven months old, who died under my care at the Boston City Hospital. The gastric capacity in this case was 300 c.c. (10 ounces), which corresponds to the gastric capacity of an infant twelve months old. You will notice the shape of the stomach, which is very significant of the symptoms I shall presently describe.

You see that the lesser curvature is not much altered, while the greater curvature is very much increased. The pathological condition of the tissues is such as would be expected

from general malnutrition. In such diseases as rhachitis there is a stretching of the muscular fibres, as well as an atrophied condition of the entire gastric walls.

**SYMPTOMS.**—The symptoms of dilatation of the stomach are essentially those of chronic indigestion. Vomiting is quite frequent, and continues until the stomach has been entirely emptied, when a period of relief comes, to last until fresh irritation arises from another supply of food. Abdominal pain, flatulence, and general discomfort are prominent symptoms. Emaciation and rapid loss in weight also occur. In some cases, in young infants, convulsions may arise, apparently due to the reflex disturbance which is produced. There are usually considerable thirst and loss of appetite. When the dilatation is of a high grade, the vomiting may occur only after considerable intervals,—twenty-four to forty-eight hours,—during which time the food does not pass out through the pyloric orifice to any degree, but collects in the stomach.

If you will look at this dilated stomach (Fig. 108), you will readily understand the mechanism of these symptoms. Under normal conditions the stomach, as I have already shown you (page 85), is somewhat tubular in shape and oblique in position. The food thus easily passes through the cardiac to the pyloric orifice. In dilatation of the stomach, on the contrary, the greater curvature is so much increased and depressed below the level of the pyloric orifice that a pouch is formed. The food, collecting in this pouch as though it were at the bottom of a well, has to be practically pumped, by the contraction of the muscular walls, up to and through the pyloric orifice. The already weakened stomach thus has to perform work for which it is not fitted, and finally is relieved by spasmodic vomiting. When only the small amount of food adapted to their normal gastric capacity is given to young infants whose stomachs are dilated, a large space of empty stomach is left above the level of the liquid which has entered the stomach. This creates a feeling of emptiness and general discomfort, so that the infant appears to be hungry when, in fact, it is only suffering from the feeling of incomplete filling of the stomach.

**DIAGNOSIS.**—On inspection the abdomen is seen to be distended and tense, and on percussion to be highly tympanitic in its upper part. Succussion is not an especially valuable diagnostic sign in dilatation of the stomach. Succussion is so frequently found in many conditions, and is so likely to be confounded with that which occurs in the colon, that it cannot be relied upon. The outlines of a normal stomach when somewhat distended vary so much in infancy that the results of percussion are often very misleading. When, however, the tympanitic resonance is found to extend below the line of the umbilicus, we may suspect that we are dealing with gastric dilatation. In infancy the cardiac end of the stomach is so slightly developed that any great increase in the area of gastric percussion to the left is an important aid in making the diagnosis.

The differential diagnosis is to be made chiefly from dilatation of the colon. In many cases when the colon is dilated it is impossible to deter-

mine whether the stomach is also dilated, since under these circumstances the colon can almost completely cover a largely dilated stomach. In older children, in cases where the diagnosis is uncertain I have found a valuable means of determining the presence of dilatation to be artificial distention. This can be done without harm or discomfort to the child by giving it first one half of a seidlitz powder and then the other half, so as to allow the chemical combination to take place in the stomach. Except in certain cases where it is very necessary to determine whether the stomach is really dilated, this is not a procedure which I am in the habit of adopting. In most cases in infants and children clinically satisfactory results can be obtained by percussion.

**PROGNOSIS.**—If the dilatation is due to congenital stenosis of the pylorus the prognosis is very unfavorable. In other cases the prognosis depends upon whether the condition arises from improper amounts of food or from some disease, such as rhachitis. In the former class the prognosis is good, and the stomach under a proper regulation of the diet soon resumes its natural size. In the second class it is not so good, and, as a rule, the stomach will remain more or less distended until the disease which causes the dilatation has been cured.

**TREATMENT.**—If the case is an obstinate one, lavage is an important part of the treatment. In many cases, however, good results are obtained simply by regulating the quality and quantity of the food. In both infants and children carefully modified milk is the food from which the best results are obtained. When the food is first given in the proper amount it will, as I have just told you, not fill the stomach nor satisfy the demands of the infant. Under these circumstances the infant will be very restless, and will often cry almost continuously from the time of one feeding until the next. You must impress upon the nurse that these signs of discomfort are liable to last for a number of days, until the stomach has more nearly resumed its normal size, and that an additional supply of food must not be given to it.

I shall report to you the case of an infant (Case 411), four months old, which illustrates dilatation of the stomach as it occurs in the first year of life. This infant, a male, was well and strong at birth. It was not nursed, but was fed on a mixture of milk, cream, and water. It was an unusually vigorous infant, and is reported to have never been satisfied with the small quantities of food suitable to its age. When it was three weeks old it was given 150 to 180 c.c. (5 or 6 ounces) at each meal. Somewhat later, in its second and third months, it gradually developed symptoms of indigestion, and when I was called to see it was in a very serious condition. It had been having frequent and prolonged convulsions. At times when it was in the convulsions it would fall into a state of collapse, the pallor of its face would be extreme, and it would look as though it were dying. On examination, nothing abnormal was found in the thorax. The entire abdomen was found to be distended, especially in the upper part, where the gastric tympany was pronounced and easily marked out by percussion. The percussion showed the stomach to be dilated, and to extend below the line of the umbilicus and far to the left of the median line.

The infant was given small amounts of food at frequent intervals. For the first two or three days it cried and screamed for more food, but the convulsions ceased, its general condition improved, and by the end of the week the distention of the stomach had subsided



very markedly and the infant had become tranquil. From this time there was no recurrence of the symptoms.

I have here in the wards to show you a colored boy (Case 412), six years old.

CASE 412.



Dilatation of stomach. Age, 6 years.

This child is markedly rhachitic. He is reported to have been in fair health, though delicate, until one month ago, when he began to have persistent vomiting. He has lost greatly in weight, has been very restless at night, and has had continual borborygmi.

Physical examination shows marked abdominal enlargement. On percussion the gastric tympany is found to extend downward as far as the umbilicus, 7.8 cm. (3 inches) to the right of the median line and 10.4 cm. (4 inches) to the left. I have marked this percussion line, which represents the greater curvature of the stomach, with spots. As the resonance of the colon is also exaggerated in this case, and as its differentiation from that of the stomach is somewhat difficult, because it evidently overlaps the lower border of the stomach, I shall endeavor to eliminate this obstacle to diagnosis by mechanical means. You see that the child readily takes half of this seidlitz powder which has been dissolved in water. The other half, which has also been dissolved in water, is next swallowed. As the combination of the two salts takes place in the stomach you can easily hear with the stethoscope the chemical action which is resulting. The child shows no signs of discomfort, and says that he does not feel any pain or any more tenderness in the epigastrium than before the powders were taken. The outline of the upper part of the stomach can now be fairly well seen, and on percussion the line of the greater curvature is found to be 2.5 cm. (1 inch) below the

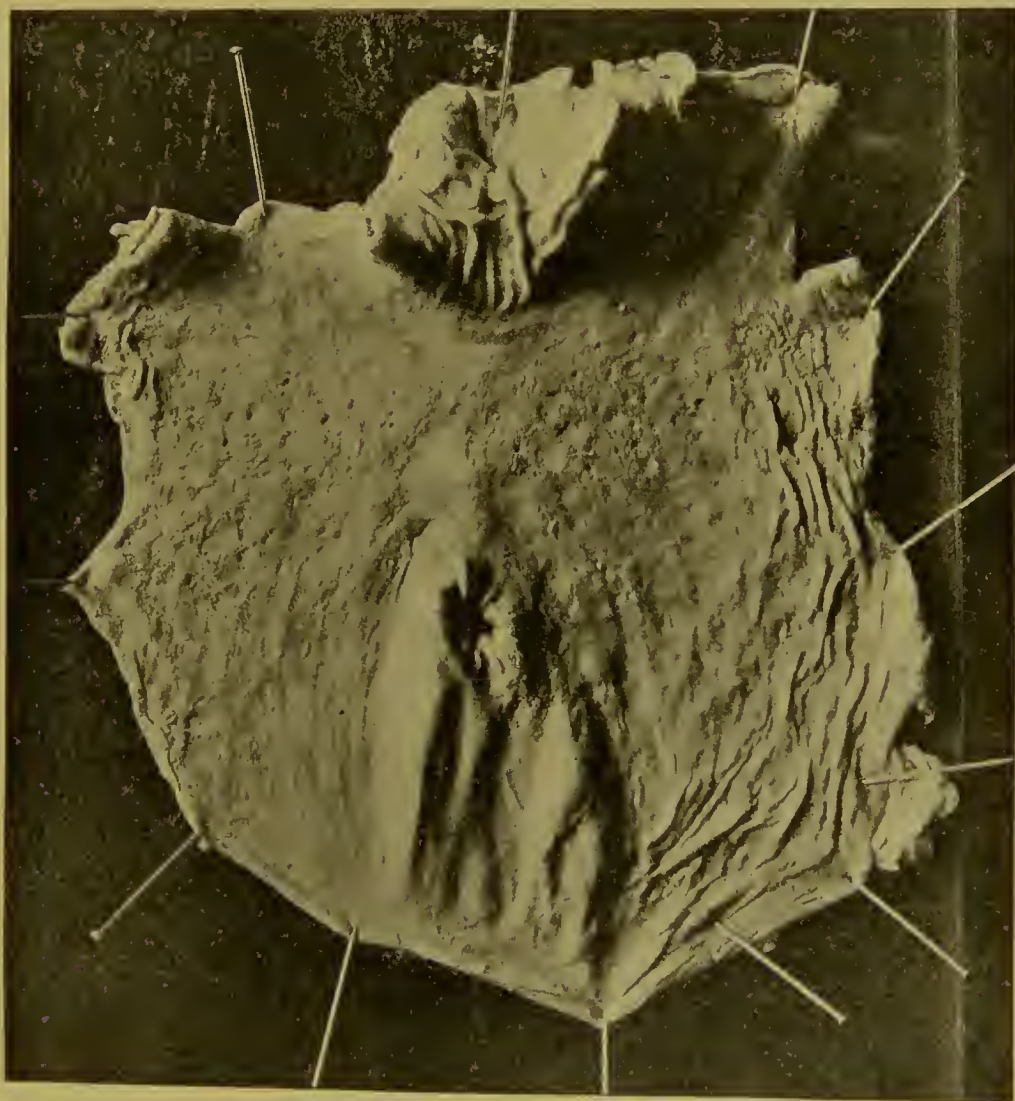
line of the umbilicus, the colon having been pushed out of the way by the distended stomach. I have indicated the line of greater curvature by a broad white line, and in this way we determine that the stomach is really dilated.

(Subsequent history.) In this case it was not found necessary to wash out the stomach more than once or twice, for as soon as small amounts of food were given at frequent intervals the vomiting ceased and the stomach gradually resumed its normal size. At the end of two months the child left the hospital free from any abnormal gastric symptoms.

**Ulcers.**—Ulcers of the stomach in infancy and early childhood are very rare, but cases have been reported. They may be non-inflammatory or inflammatory, the distinction between the two often being very difficult to make.

Through the kindness of Dr. Northrup I am enabled to report to you such a case, occurring in a female one year old who was under his care. I also have here the stomach (Fig. 109) to show you.

FIG. 109.



Follicular ulceration of stomach. Female, 1 year old.

The infant (Case 413) was under treatment for one month. It had vomiting and diarrhoea. During the first week that it was in the hospital its temperature varied from 38.8° to 39.4° C. (102° to 103° F.), after that being normal or subnormal. The respirations varied from 40 to 50, and the pulse from 120 to 140. In the second week it began to refuse



its food and to emaciate. The diarrhoea continued, and the vomiting was persistent. The vomitus was somewhat brownish in color. The child died of exhaustion.

On examining the stomach you see that the lining mucous membrane is covered with small ulcers, varying in size from dots to 1 cm. ( $\frac{3}{8}$  inch) in diameter. The lesions appear to be follicular ulcerations. You will notice that in the middle of the specimen is a much larger ulcer, which has perforated the gastric wall. There is no evidence of an inflammatory condition, and the cause of these lesions is unknown. There is, however, a certain degree of necrosis around the ulceration.

**New Growths.**—Morbid growths in the stomachs of infants and young children are so extremely rare that their occurrence need merely be referred to.

**INFLAMMATORY.**—The inflammatory lesions of the stomach may be either acute or chronic, and are termed gastritis.

**Acute Gastritis.**—Acute gastritis may be divided into (1) gastritis catarrhalis, (2) gastritis corrosiva, and (3) gastritis pseudo-membranosa.

Before describing these forms I must state that, in my experience, the cases in which a catarrhal condition of the stomach can be proved to exist are very limited in comparison with those in which the functional disorders which I have already described are present. I believe that in a large number of cases which are spoken of as gastritis catarrhalis no catarrhal condition is present, and that they would be much better classified under the heading of indigestion. I am led to believe this from the numerous cases in which a diagnosis of gastritis has been made during life, and in which, at the autopsy, no definite lesion has been found. When, however, gastritis is present, as a rule the acute form is more common in infants, while the chronic form is more frequent in children towards the age of puberty.

*Gastritis Catarrhalis Acuta* (Acute Gastric Catarrh).—The cause of acute gastric catarrh is somewhat obscure, but it is usually supposed to arise from an exaggerated form of indigestion, or from the presence of irritants of various kinds, among which too hot food has been cited.

**PATHOLOGY.**—The pathological lesions which characterize acute gastric catarrh are hyperæmia of the gastric mucous membrane, hypersecretion of mucus, small punctate hemorrhages, and slight thickening of the mucous coat.

Special work on this subject has been done by Epstein in Germany and by Booker in this country. According to Booker, where a catarrhal condition of the gastric mucous membrane is present the milk remains much longer in the stomach than under normal conditions,—possibly four or five hours, or even more. A microscopic examination of the gastric contents in these cases shows various micro-organisms, and sometimes epithelial and pus cells. The small number of bacteria found in cover-slip preparations from the contents of the stomach affords a most striking contrast to the large number of bacteria which, under like circumstances, are found in the feces.

**SYMPTOMS.**—Two forms of acute gastric catarrh are usually described, the division being made according to the length of the febrile period. In one form there is little or no fever, while in the other the temperature is



high. The first class, or afebrile form, is by far the more common, and is what is usually spoken of as gastritis catarrhalis. It is subacute rather than acute. According to my experience, it is difficult and almost impossible to state definitely the symptoms of the afebrile form of acute gastric catarrh. They so nearly approach those which occur in cases of indigestion, where we believe no gross pathological condition exists, that we should always be guarded in our use of the word catarrh. Pain is so common a symptom in all gastric disturbances, the existence of tenderness is so difficult to determine in infants and young children, and a hypersecretion of mucus is so often known to occur without the presence of an inflammatory condition, that there does not seem to be any one symptom upon which we can rely. The general picture of the disease which is supposed to represent acute gastric catarrh is that of fever, nausea, vomiting of food mixed with mucus and at times of mucus alone, and a sense of tenderness, uneasiness, and discomfort in the epigastrium. There may be frontal headache, a rather swollen, coated tongue of somewhat glassy appearance, and often a slight follicular pharyngitis. There is loss of appetite, with, at times, hic-cough and eructations of gas. The bowels are usually constipated at first, but after three or four days diarrhoea may result.

Where the infant or child seems prostrated for a few days, and sick beyond what would naturally be expected in an acute attack of indigestion, and where, in combination with a somewhat heightened temperature, frequent vomiting of mucus occurs, we are justified in supposing that we are dealing with a catarrhal condition.

TREATMENT.—The treatment of cases of this kind is the same as that which I have described in speaking of indigestion. Food should be withheld from the stomach for many hours, for, as I have just told you in speaking of the pathological conditions which occur in gastritis catarrhalis, the food remains so long in the stomach that a fresh supply at short intervals will act as an additional source of irritation. In those cases which do not respond readily to long intervals of rest and to feeding with small quantities of a modified alkaline milk, lavage will prove of value. Much judgment should be used as to the time when the food is to be increased in strength, for unless great precautions are taken relapses will frequently occur, and as a result the disease may finally become chronic. After convalescence has been established the child will begin to gain in weight. Some simple tonic, such as *nux vomica*, is usually indicated for a week or ten days until the child has recovered its strength. During the beginning of the attack, when food is being withheld, if the child is made very restless by extreme thirst, teaspoonful doses of iced soda water can be given, but with caution and as seldom as possible. The second or febrile form of acute gastric catarrh is rare, but is of much more serious import than that of which I have just spoken. It is characterized by high fever,  $39.4^{\circ}$ ,  $40^{\circ}$ ,  $40.5^{\circ}$  C. ( $103^{\circ}$ ,  $104^{\circ}$ ,  $105^{\circ}$  F.). The invasion is very acute. It may last for two or three weeks and show severe and alarming symptoms. There may be

active vomiting, delirium, and sopor in the beginning, so that it will be impossible to determine whether or not one of the other acute febrile diseases is developing. The characteristic symptoms of gastric catarrh develop later, and then the differential diagnosis is easily made. Instead of the cessation of the vomiting in the first twenty-four or forty-eight hours, as in scarlet fever, and of the continuance of the cerebral symptoms, as in meningitis, or of the development of pulmonary symptoms, as in pneumonia, the vomiting continues, though not quite so frequent as in the beginning, the mind becomes clear, and the symptoms point to the abdomen rather than to the head or the thorax. The onset of pneumonia in some cases, though in my experience rarely, simulates this disease. The pulse is rather irregular. There is usually constipation at first, followed by diarrhœa.

The prognosis is good, except in very debilitated children.

The child should be placed in a darkened room, soothing applications applied to the abdomen, and small quantities of iced soda-water given. The food should be given as I have just described in the other form of gastritis catarrhalis; that is, in very limited quantity and at long intervals. If there is much exhaustion, stimulants are indicated.

*Gastritis Corrosiva Acuta.*—Corrosive lesions of the mucous membrane of the stomach are at times produced by swallowing irritants, such as arsenic, carbolic acid, and caustic fluids. In these cases the lesions are usually found on the summits of the rugæ.

The treatment is by washing out the stomach with large quantities of water, administering the proper antidote, and feeding the child on a liquid diet so modified as to be as little irritating as possible to the injured mucous membrane.

*Gastritis Pseudo-membranosa.*—The membranous form of gastritis is extremely rare in infancy and childhood. Cases have been reported, notably those of Wollstein. In these cases the congestion of the rugæ was very marked, and along the greater curvature extended over an area of a number of inches. There was a thick grayish-green membrane, with some erosions. The gastric walls were much thickened.

The *symptoms* of gastric disturbance in these cases are often almost entirely absent, but there may be vomiting, pain, and tenderness in the epigastric region, and insatiable thirst. A pathognomonic symptom would be the vomiting of shreds of membrane, with or without an admixture of blood. This symptom is, however, extremely rare, because the membrane is usually adherent, so that a differential diagnosis is often impossible.

The *prognosis* is very unfavorable, and the *treatment* is purely symptomatic.

*Gastritis Catarrhalis Chronica* (Chronic Gastric Catarrh).—Chronic gastric catarrh, as I have already stated, is not usually met with in infancy, but occurs in later childhood. It is especially common in the summer months, and is generally the result of neglect or of improper treatment of the acute form of the disease.

**PATHOLOGY.**—The pathological condition which is found in chronic gastric catarrh is the result of long-continued hyperæmia. There is often a slaty discoloration of the mucous membrane, with cellular infiltration of the submucosa. In addition to this there is usually found a considerable quantity of tough mucus.

**SYMPTOMS.**—The symptoms are not so clearly defined as in the acute form of the disease, but are variable and of a rather sluggish type. The tongue is apt to be much coated and the breath to have a disagreeable odor. There is considerable abdominal distention after meals, so that the children complain that their clothes feel uncomfortable.

Frontal headache is apt to occur. The children gradually grow thin and anæmic. They vomit at irregular intervals, and are usually constipated. There is often a slight cough, and the symptoms, so far as the stomach is concerned, may form so small a part of the general picture of the disease that the child is not infrequently brought to the physician on account of its cough and because it is supposed to have some pulmonary affection.

**PROGNOSIS.**—Although the disease is often somewhat intractable, the prognosis under proper treatment is good. It may last for three or four months; but in many cases which are usually considered chronic gastric catarrh it has seemed to me there is no organic lesion, but that the disease is functional in its character, and the prognosis consequently very good.

**TREATMENT.**—It is often necessary in these cases to precede the treatment by carefully washing out the stomach. We must remember, however, that a considerable quantity of mucus may be in the stomach which cannot be removed by washing, so that if the symptoms continue after one or two washings, even though no mucus is returned by the tube, we should repeat this treatment from time to time. The diet should be an alkaline modified milk, with a low percentage of proteids, if necessary peptonized, and a moderate percentage of fat and sugar. The percentages of the different elements should be increased as improvement in the gastric symptoms takes place, and later broths and milk can be tried. Symptomatically in certain cases pepsin, dilute hydrochloric acid, and bismuth are occasionally indicated. A valuable tonic in the after-treatment of these cases is *nux vomica*.



## LECTURE XLV.

## DISEASES OF THE INTESTINE.

DISEASES of the intestine may be divided into three classes,—developmental, functional, and organic.

**DEVELOPMENTAL.**—Certain malformations and malpositions of the intestine occur as a result of abnormal development. The malformations are of that class which I have described in a previous lecture when speaking of Meckel's diverticulum and of imperforate rectum (pages 426–433). Malpositions are met with in infants where there is a transposition of the abdominal organs.

**DIARRHŒA.**—As vomiting is the most significant symptom of gastric disturbance, so diarrhœa resulting from increased intestinal peristalsis is the most characteristic symptom of intestinal disturbance. Diarrhœa is always a symptom, never a disease. There seems to be a predisposition to diarrhœa in the first two years of life, which decidedly lessens as the child grows older. The most frequent time for the occurrence of diarrhœa is during the summer months.

**PROPHYLAXIS.**—Much can be done at all seasons of the year to prevent the occurrence of diarrhœa, but prophylaxis is of the utmost importance in warm weather. The children should be protected by proper clothing from extremes of heat and cold, and from dampness. They should, if possible, be taken away from crowded or unclean districts in cities and towns during the hot weather, and have the advantages of fresh country or sea air and good hygienic surroundings. Both the quality and the quantity of the food should be carefully regulated. The milk and the water should be pure and sterile, and in very hot weather an extra amount of water should be allowed and the solid food somewhat diminished in amount. Uncooked fruits and food are contra-indicated in very hot weather. Especial attention should be paid to any slight indisposition which may arise in hot weather, as it may render the child more vulnerable to the various causes of diarrhœa.

**INTESTINAL CONTENTS.**—Before speaking in detail of the various diseases of the intestine, I shall describe to you some of the more important abnormal appearances which are met with in its contents. These changes are significant of diseased conditions, though not necessarily of any especial disease. The intestinal contents should be studied in regard to their color, consistency, composition, odor, and amount.

**COLOR.**—I have already described to you (page 117) the normal appearances of the fecal discharges, and I shall now show you some that are abnormal.

This specimen, which is numbered 16 (Plate III., facing page 112), is what is usually spoken of as clay-colored. This clay color may be due to a diminution in the amount of bile which enters the intestine, or to undigested fat. This color is abnormal, and is usually met with in intestinal diseases of a subacute or a chronic type. It does not necessarily indicate a serious condition, however, as even a small plug of mucus may interfere with the flow of bile into the duodenum.

This specimen, which is numbered 17, is the light green color, which may be simply a change that has taken place after the fæces have been passed, and which often is not significant of any especial pathological condition. It may, however, show that the changes which have taken place in the food during its passage through the intestine have not been entirely normal. It is the least important of the changes which take place in the color of the intestinal contents. The colors in these next two specimens, numbered 18 and 19, are what may be seen in a more serious disturbance of the enteric tract. These colors may appear in any of the intestinal diseases which are accompanied by diarrhœa, but are significant of no especial disease. They are merely to be considered pathological in contradistinction to the normal colors in these other specimens, 3, 4, 6, 7, 8, 9, and the beginning abnormal condition represented in 17.

Besides these shades of green there are a great many varieties of color produced by the mixture of green, yellow, white, and brown. These are valuable merely as instructing us whether we are dealing with a normal or an abnormal condition of the intestinal contents, and, as I have already told you in my general remarks on diagnosis, are not significant of any one disease, either functional or organic. Much variety in the color also arises from the admixture of blood, mucus, and shreds of membrane. In this connection it is well to remember that the yellowish-white lumps seen in undigested fæces are often made up of fat as well as of proteid material.

The color of the intestinal contents may also be changed by the administration of various drugs, such as iron, which causes a more or less black color. Bismuth gives the colors which you see in these three specimens numbered 12, 13, and 14. Number 12 is the color which was produced by giving to an infant 0.18 gramme (3 grains) of bismuth every two hours for six doses; number 13, where 0.24 gramme (4 grains) of bismuth was given every two hours for six doses; and number 14, where the latter dose had been omitted for twenty-four hours. The size of the dose and the intervals between its administration will of course produce different shades of color.

Where the solids of the intestinal contents are much reduced in proportion to the serum, as in cases of acute and frequent diarrhœa, the discharges become more and more fluid, and sometimes almost entirely lose their color and look like water.

CONSISTENCY.—In the first year of life, or while the infant is having only milk for its food, the consistency of the fæcal discharges is inter-



mediate between solid and fluid, and the discharge, as a rule, is smooth and free from lumps. As the infant begins to take other forms of food and a mixed diet, the faecal discharges gradually become more solid. The consistency of the faecal discharge is abnormal when it becomes liquid, as in diarrhœa, or when it is too solid, as in constipation.

**COMPOSITION.**—In addition to the various substances which make up the food which enters the intestine, the faecal discharges contain bile, mucus, epithelial remains, and many bacteria. In diseased conditions they may also contain certain morbid elements, such as blood, pus, and membrane. In intestinal diseases of both an acute and a chronic type the mucus may be very largely increased, but it cannot be considered to be especially characteristic of an inflammatory condition, as the secretion of mucus apparently may be very much increased in purely functional conditions. The bacteria are very numerous and of many varieties, but in most cases the detection of any especial form of these organisms does not aid us in diagnosing the especial disease. Notable exceptions to this statement are where one finds the typhoid bacillus, the comma bacillus, and the amœba coli.

**ODOR.**—While in the normal faecal discharges of infants fed entirely on milk the odor is comparatively slight, it becomes much stronger as other articles of food, either of a starchy or of a proteid nature, are added to it. Where an abnormal condition exists, various changes take place, as in acid fermentation, where the odor is sour, and in albuminous decomposition, where the odor is very foul. Although these conditions can scarcely as yet be considered of great diagnostic importance, they are sufficiently so for us to make use of them in the diagnosis and treatment of intestinal diseases. Thus, where acid fermentation is supposed to be present, a reduction in the percentage of the sugar, and perhaps of the fat, is indicated, while where albuminous decomposition is suspected a reduction of the proteids in the food is called for.

**AMOUNT.**—In estimating the amount of the faecal discharges we must consider the total amount in twenty-four hours, and not the large or small amount which may occur at one movement. The total amount in twenty-four hours is of much importance in both the acute and the chronic diseases of the intestine. In the acute diseases, the more frequent the diarrhœa and the larger the amount the greater is the exhaustion and the worse is the prognosis. In some chronic diseases the total amount of faecal discharges may be very large. In these cases the larger the total amount the less has been the absorption and the worse is the prognosis, for this condition is an indication that the child is being starved from a lack of power to absorb the food which has been given to it.

**FUNCTIONAL.**—The functional diseases of the intestine may be classed as acute, chronic, and eliminative.

**ACUTE (Simple Diarrhœa).**—The acute functional disturbances of the intestine may be of nervous origin, or they may arise from intestinal indigestion.



**Nervous.**—In certain infants and children whose nervous system is easily affected exaggerated peristalsis causing diarrhœa may arise from a number of causes without any known lesions, fever, or gastric disease. Among these causes may be cited heat, cold, and fright. In like manner in these individuals foreign bodies, food or otherwise, may by simple reflex irritation cause such a nervous disturbance as to produce diarrhœa. In these cases either the small or the large intestine, or both, may be affected, and, so far as we know, the mucous membrane is either normal or simply hyperæmic. There is more or less serous exudation. These cases are rare in comparison with the other forms of diarrhœa, such as those which are caused by bacteria, and in them intestinal decomposition and intestinal inflammation are not present primarily.

**SYMPTOMS.**—The symptoms of simple diarrhœa are very apt to appear suddenly. There is usually abdominal pain, not, as a rule, of great intensity. At first there are two or three rather liquid yellowish-brown discharges occurring at intervals of perhaps one-half or one hour, and often accompanied by considerable flatus. There is a certain amount of restlessness, pallor, and exhaustion. Vomiting is rare. The temperature, as a rule, is not raised, or is raised very slightly. The pulse is rather weak and somewhat quickened. The number of the discharges may be eight, ten, or twelve in the twenty-four hours, and these soon become watery and of a lighter color, but are seldom green. The odor is somewhat increased, but not excessively. These symptoms, unless they are exaggerated by improper food or by bad treatment, usually disappear in a few days.

**TREATMENT.**—If there is a known cause, such as some intestinal irritant, a dose of castor oil or calomel may be given, but, as a rule, this is not necessary. The child should be kept in bed. Food should be withheld for some hours. A few drops of tinctura opii camphorata and hot applications to the abdomen are indicated for pain. If there is a tendency for the diarrhœa to continue, the subnitrate of bismuth may be given, and in some cases where there are signs of exhaustion a stimulant may be needed. For several days the diet should be simply milk heated for twenty minutes at 75° C. (167° F.) and containing ten per cent. of lime water.

I must impress upon you that these simple diarrhœas, especially in hot weather, should never be allowed to continue, as they render the intestine vulnerable to the more serious diseases, which may at any moment gain an entrance in this way.

**Tubular.**—In addition to these more common intestinal affections of nervous origin is one that is called *tubular*. This disease is so rare before the age of puberty that it need only be alluded to. It is a condition of the mucous membrane of any part of the intestine in which an exudation of mucus takes place in such a way that masses closely simulating a membrane may form on the surface. When discharged through the rectum they are sometimes found to have formed a cast of the intestine. These

masses are mostly made up of mucus, and may occur in shreds of greater or less extent as well as in the tubular form.

The disease is supposed to be of nervous origin. The symptoms are pain, tenderness, and tenesmus. The temperature is usually normal.

The *prognosis*, as a rule, is good, although in some cases the disease may be much prolonged.

The *treatment* is to be directed essentially to improving the general health and the nervous condition, the local treatment being merely symptomatic.

**Indigestion.**—Disturbances arising from intestinal indigestion in most cases may be located in the duodenum. It has yet to be proved that any pathological lesion is present in these cases, and for the present they must be looked upon as functional.

These cases vary in their symptoms with the individual and according to the part of the duodenum which is most involved. In one set of cases the disturbance of digestion is shown simply by the increased peristalsis, such as I have just described in the nervous cases, but here the evidence points to an undigested condition of improper articles of food which have been given and which appear in the discharges.

The treatment of this class of cases is very simple, and consists in first giving a laxative and then regulating the diet according to the age of the individual.

What is usually spoken of as a "bilious attack" represents another class of cases. This condition is very rare in infancy, and usually occurs in middle and later childhood. In these cases, in addition to the increased peristalsis and evidence of undigested food, there are frequently icterus and vomiting of bile. In addition to these symptoms there may be headache and excessive nausea. The icterus is usually slight in degree, but often is marked and is noticeable in the conjunctivæ and in the urine. Here is a specimen (Plate III., facing page 112, No. 11) which I have numbered 11, and which shows the staining of bile on the napkin of an infant during an acute attack of indigestion involving the duodenum and accompanied by icterus. The temperature in these cases is usually slightly raised for a few days and then becomes subnormal. In a certain number of cases the faecal discharges become clay-colored. This color is often produced mechanically, as I have already explained.

Although the symptoms in these acute attacks may often appear quite serious, the prognosis is always good. In some individuals they are liable to recur even when the diet and the general health are well attended to.

In the treatment of this class of duodenal disturbances we must consider that the fats in the food are in all probability especially liable to prolong the disease by not being properly digested so long as the function of the duodenum is involved. We should, therefore, in treating these cases, lessen the amount of fat given in the food. I have found that the treatment which most speedily shortens the attack is (1) total restriction from food



for twelve hours, with the administration of small quantities of cold, sterilized water if the thirst is excessive, and (2) the administration of small quantities of milk modified as in this prescription (Prescription 77):

PRESCRIPTION 77.

Fat . . . . .	0.16
Sugar . . . . .	6.00
Proteids . . . . .	3.00
Lime water . . . . .	10.00

The mixture to be heated to 75° C. (167° F.) for twenty minutes; from 120 to 180 c.c. (from 4 to 6 ounces), according to the age, to be given every three hours.

Under this treatment the icterus usually passes away in a few days, and the child can then soon be given its ordinary food.

**CHRONIC.**—The chronic functional diseases of the intestine may be the result of acute nervous disturbances, or they may arise from a number of prolonged attacks of acute indigestion. Incontinence of fæces and constipation are also forms of chronic functional intestinal disturbances. Under this same heading we can class infantile atrophy.

**Nervous.**—In the chronic form of nervous functional intestinal disturbance either the small or the large intestine may be affected, and, as I have already stated in describing the acute form, the condition of the mucous membrane, so far as we know, is either normal or hyperæmic. The causes are the same as in the acute form. This class of cases is not especially common, as they are merely a prolongation of the symptoms which I have already sufficiently described in speaking of the acute cases. The treatment of these cases is essentially with stimulants and care of the general health.

**Indigestion.**—The chronic form of functional intestinal indigestion plays an important part in intestinal diseases, especially when it is located in the duodenum. Chronic indigestion of the duodenum constitutes a disease of itself, and is one of the most difficult to cure which we meet with. It has usually been spoken of under the names of chronic gastro-duodenal catarrh and mucous disease. We at present, however, have no proof that either a catarrhal or any other pathological lesion of the mucous membrane is present in these cases, and the weight of evidence is in favor of the view that the disease is purely functional.

**ETIOLOGY.**—The etiology of chronic duodenal indigestion is in many cases obscure, but in a large number of cases it is produced by the continual administration of food which is not adapted to the age or digestive capabilities of the child. It is at times met with as a sequela of some exhausting disease, such as typhoid fever, pneumonia, or one of the acute exanthemata. It very rarely occurs in early infancy, being usually met with during the middle and later periods of childhood.

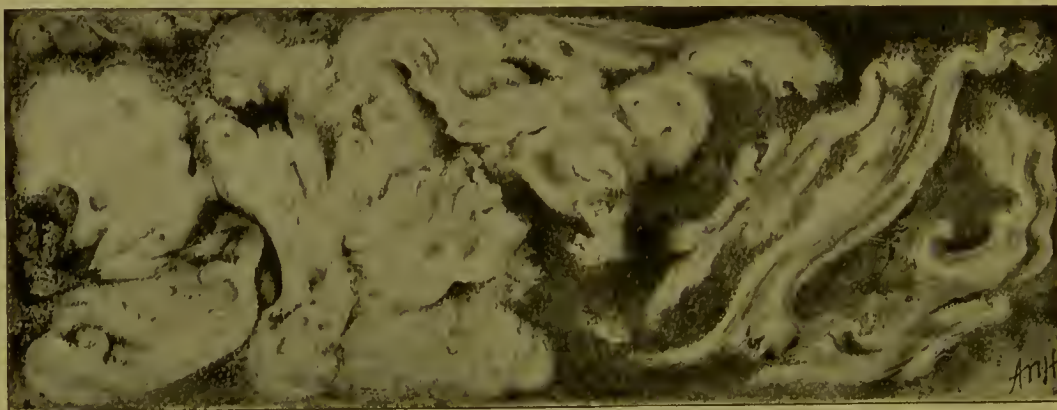
**SYMPTOMS.**—The symptoms are at first somewhat varied. The disease



may be preceded by a number of attacks of gastro-enteric indigestion of a subacute character. A tendency to nausea and vomiting extending over a number of months may sometimes precede the full development of the disease. The gastric disturbance, however, is not marked, and is probably a reflex condition depending upon the functional disturbance of the duodenum. At first the fecal discharges show merely the various changes which occur in ordinary indigestion, sometimes manifesting a tendency to diarrhoea and sometimes to constipation. The color of the discharges at this early period is not significant of anything beyond ordinary indigestion, and is usually a mixture of yellow, white, and green. As the disease progresses, certain characteristic symptoms arise and definitely mark its presence. Mucus begins to appear in the fecal discharges, and soon becomes quite large in amount.

I have here a specimen (Fig. 110) of the shreds and masses of mucus which appear in the discharges, and which, in combination with the other symptoms which I am about to describe, are so significant of the disease that it will be well for you to examine them closely.

FIG. 110.



Mucus from fecal discharges in a case of chronic duodenal indigestion.

With this hypersecretion of mucus, which I shall again impress upon you is not necessarily an indication of an inflammatory condition, the child begins to be fretful, to be wakeful at night, to grind its teeth, and to lose in weight. The skin becomes dry, and there is usually a coexisting follicular pharyngitis which causes a short, dry cough. The child gets tired easily, and complains of pain in the epigastrium after eating. The abdomen is apt to be distended and tympanitic. There are frequently frontal headache, a coated tongue, and a disagreeable odor to the breath. The fecal movements now begin to become clay-colored, and the skin to assume a sallow tint, with at times a slight amount of icterus. Sometimes an exacerbation of all the symptoms takes place, resulting in an acute attack of indigestion. These symptoms, varying in intensity, and sometimes ceasing to be prominent for days or weeks, usually continue for months, and in intractable cases may last for years. The temperature in this disease is usually normal, sometimes subnormal, but may of course, where an exacerbation occurs, be

somewhat raised. The pulse is usually moderately slow. Sometimes a subacute form of bronchial catarrh accompanies the disease, but it does not appear to be a part of it. There is often a craving for large quantities of sugar.

**DIAGNOSIS.**—When all the symptoms are present, the diagnosis of chronic duodenal indigestion is not difficult. The appearance of the child is characteristic. Its eyes are dull and heavy; its skin is dry and harsh and sometimes slightly icteric, while the loss of flesh, the distended and tympanitic abdomen, and the coated tongue are more marked than in any other disease. Where, in addition to this picture, an examination of the fecal discharges shows them to be clay-colored and to contain a large amount of mucus, the diagnosis is quite evident. The disease which is most commonly mistaken for chronic duodenal indigestion is pulmonary tuberculosis. The short, dry cough, the emaciation, and in some cases the bronchial catarrh, often make parents and physician fear that this serious disease is present. If, however, the entire history of the case is studied carefully, pulmonary tuberculosis can soon be eliminated.

**PROGNOSIS.**—The prognosis of chronic duodenal indigestion is in most cases good. Even in those cases which last for a period of years the health is usually entirely restored. Where, however, the disease has lasted for a long time, and the child is in a very debilitated condition, the prognosis becomes more serious.

**TREATMENT.**—The treatment of this disease is essentially by diet, and not by drugs. Such articles of food should be given as will be chiefly digested by the stomach and will not tax the duodenal digestion. This of course indicates a proteid diet, and contra-indicates the administration of starches, sugars, and fats. In order not to tax the disturbed duodenum by overloading it in its weak condition, small amounts of food at shorter intervals than usual are found to produce a better result than the regular three or four daily meals. The diet which I have found most valuable in treating these cases is a milk so modified as to have a low percentage of sugar and fat, a high percentage of proteids, and ten or fifteen per cent. of lime water. Soups of various kinds, and meat, can also be given, and the crust of French bread in limited quantity. A valuable adjuvant to this treatment, as a mild astringent and stimulant, is a small amount of claret, preferably given in seltzer water. The meals should be five in the twenty-four hours. It is exceedingly difficult in most cases to keep the child on this diet, but if it is rigorously enforced the duration of the disease will be decidedly shortened. As the epigastric pain and the amount of mucus in the discharges grow less, the diet may be somewhat varied by giving fish and eggs, and the percentage of lime water in the milk may be reduced to five. As relapses occur very easily, however, it is generally best to continue with this rigid diet until the fecal discharges have become normal in color and have not shown the presence of mucus for a number of weeks. In mild cases where there is much constipation, small doses of



calomel, or any mild laxative, are indicated. Podophyllin can also be given, as in this prescription (Prescription 78):

## PRESCRIPTION 78.

<i>Metric.</i>		<i>Apothecary.</i>	
	Grammæ.		
℞ Podophyllin. . . . .	0   06	℞ Podophyllin. . . . .	gr. i;
Alcohol. . . . .	3   75	Alcohol. . . . .	ʒi.
M.		M.	

Sig.—From 3 to 5 drops, according to the age of the child, in the morning and evening, lessening the dose if it causes more than two discharges daily.

Where there is a tendency to diarrhœa, small doses of bismuth are found to be valuable.

Tincture of nux vomica freely diluted in water and given in doses of a few drops after each meal seems in some cases to be of value.

The remainder of the treatment is essentially symptomatic, and if the children are weak and anæmic tartrate of iron and potassium can be given.

During the whole course of this disease cod-liver oil is contra-indicated, but where the disease has been cured and the child is left weak and emaciated it may in some cases be beneficial. Its administration, however, should always be carefully supervised, as it may cause a relapse.

I have here a child (Case 413*a*), three years old, who has an attack of chronic duodenal indigestion, and who represents very well the general picture of this disease. I wish you to notice especially in this case the distended abdomen, the dry, harsh skin, which is slightly icteric, and the evident loss of flesh. She has been affected by the disease for the past two months. The prominent symptoms have been epigastric pain and clay-colored movements with a hypersecretion of intestinal mucus.

This little girl (Case 414), six years old, represents also a case of chronic duodenal indigestion.

She was healthy at birth, and was nursed until she was thirteen months old. During her first year she had an attack of bronchitis, and since then she has been subject to cough. Previous to this attack she has never had any intestinal disturbance. Her abdomen is said to have been always rather prominent. From her earliest infancy she has been a nervous child, has not slept well, has talked much in her sleep, and has occasionally walked in her sleep. Six months ago she began to lose in weight, and two months ago her cough became quite severe. She then had an attack characterized by vomiting for twenty-four hours, followed by anorexia, fever, languor, and apathy; the bowels became constipated, the skin icteric, the urine dark-colored, and the fecal movements light-colored. She had an intense craving for sugar, and ate all that she could lay her hands on, so that she had to be watched very closely to prevent her satisfying this morbid appetite. Her appetite for other articles of food was poor. You will notice that the abdomen is distended and tympanitic, and that the tongue is coated. The breath has a disagreeable odor, and there is loss of flesh. There is a follicular pharyngitis, which is evidently the cause of the cough, as nothing abnormal can be detected in the lung or the nose.

(Subsequent history.) The child was placed on the following diet. Her first meal was milk so modified as to contain fat 2, sugar 3, proteids 4, lime water 10. With this meal she was allowed to have a small amount of the crust of French bread. The second meal consisted of broth and the crust of French bread, and one ounce of claret in half a tumbler of seltzer water. The third meal consisted of meat, the crust of French bread, claret, and seltzer water; the fourth, of soup, the crust of French bread, claret, and seltzer water; the



fifth, of the modified milk and the crust of French bread. After each meal three drops of tincture of *nux vomica* were administered.

This diet was carried out rigorously for one week. At the end of that time the child looked much better, the urine was clear, the faecal movements began to resume a more natural color, the mucus in the discharges was very much lessened, and the abdomen was not

CASE 414.



Chronic duodenal indigestion. Female, 6 years old.

so much distended. The tongue was less coated, and the cough had almost disappeared. It was also found that the craving for sugar had much decreased. The diet was then slightly increased in variety. At the end of a month the child had recovered entirely, and some weeks later an ordinary diet at the usual times was given to her.

**Incontinence of Fæces.**—Incontinence of fæces is a condition in which there is a loss of power of the sphincter to control the movements. It may be due to organic or to functional causes. The organic causes are very rare in childhood, and will best be spoken of in connection with the diseases in which they occur. Functional incontinence may arise from nervous influences, such as excessive mental fatigue, or from stretching of the rectum from habitual constipation.

This boy (Case 415), eleven years old, represents the nervous type of the disease. He has been much overworked at school, has been made to study a number of languages, and has been allowed to take only a very limited amount of exercise in the open air. He has completely lost control of the sphincter ani, and, as you see, is very anæmic and weak. Nothing abnormal has been found on a physical examination.

(Subsequent history.) The boy was taken from school, relieved entirely from his studies, and kept in the open air most of the day. Under this treatment, in addition to the administration of tartrate of iron and potassium and claret, he improved rapidly, and in two months was entirely well.

This boy (Case 416), seven years old, came to the Children's Hospital, during the service of Dr. Lovett, with a history of incontinence of fæces lasting over a year. He illustrates the condition of incontinence from habitual constipation, as the incontinence was found to depend on stretching of the rectum by impacted fæces.

The rectum was emptied by a dose of castor oil and an enema each day. At the end of a week the boy had ceased to have involuntary fæcal movements, and he has since continued well.

**Constipation.**—By constipation is meant a condition in which the movements of the bowels do not take place as often as is normal for the individual, and in which the consistency is abnormally increased. Constipation is a symptom, and not a disease. It is a relative term, as what would be normal in one individual may be abnormal in another. During the first year of life two or three daily discharges may be considered normal; in the second year two discharges; and in the third and fourth years one discharge is the usual number. The causes of constipation are varied, and in many cases rather obscure. Mechanical obstruction may produce constipation. Thus, as the sigmoid flexure is proportionately long in infancy, flexions may occur, with resulting obstruction. The usual cause of constipation, however, is of a functional character, and may be *spasmodic* or *atonic*.

*Spasmodic.*—The spasmodic cases are rare, but should be recognized, as they frequently cause much disturbance of the child's general health. In these cases the fæcal movements are usually much increased in size and consistency. This condition produces so much pain and irritation in the rectum that the child endeavors not to have a movement.

*Atonic.*—The atonic is the most common form of constipation, and simply represents a sluggish condition of the intestinal peristalsis. It is usually caused by food which is not adapted to the digestion of the special child. Thus, in some cases cereals, such as oatmeal, seem to produce this condition, although in a large number of cases they relieve it.

As a rule, constipation can be easily cured, but some cases are extremely intractable and last for a number of years. When the intestine has become more developed and assumed the relative proportions found in adult life, the constipation is very apt to pass away, so that we may in almost every case give a favorable prognosis. Constipation can usually be cured by strict attention to the regulation of the diet by the use of fruits, vegetables, and cereals. In young infants an increase of the fat in the milk will in quite a number of cases relieve it. Variation in the percentage of sugar is occasionally found to be efficient in curing the constipa-

tion. Many drugs have been employed in the treatment of constipation, but, as a rule, we should endeavor not to use them, as they are very apt to be only temporary in their action. In connection with the diet, I place most reliance upon enemata and laxative suppositories, such as those made of glycerin or of gluten. Massage of the abdomen is useful in some cases.

**Infantile Atrophy** (Marasmus. Athrepsia).—Infantile atrophy is essentially a disease of infancy and early childhood. It is a condition in which extreme atrophy of all the muscular tissues takes place without demonstrable disease of any of the organs. It is apparently due to a vice of absorption, although this has by no means been clearly proved.

The primary cause of infantile atrophy is unknown. In a number of cases the disease seems to be secondary to grave intestinal disturbances, whether of toxic or of organic origin.

**PATHOLOGY.**—The pathological conditions which are found in cases of infantile atrophy are exceedingly unsatisfactory, and have not given us much information concerning the disease. There is an atrophic condition of all the muscles. Nothing abnormal is found in the various organs which can be especially attributed to this disease. It is supposed by some pathologists that the lymph-glands are enlarged; but this enlargement does not seem to be a prominent feature. No pathological condition of the mesenteric lymph-glands has been found, and the atrophy of the mesentery around them is so great that their increase in size may be seeming rather than real. In the intestine, although in some cases there is considerable atrophy of the mucous membrane and the submucous tissue, no characteristic lesion has been proved to be present.

**SYMPTOMS.**—The symptoms of infantile atrophy are those of starvation. The infant begins to emaciate, and extreme loss of weight is the prominent feature of the disease. The food is apparently digested well, and the fecal movements are often of a normal character; in many cases the total amount in the twenty-four hours is abnormally large. The appetite is, as a rule, lessened, the temperature is normal or subnormal, the pulse is weak, and the respirations are generally normal. Usually the infant seems not to suffer from pain, being sometimes quite apathetic, but in some cases extreme fretfulness and restlessness occur. Vomiting, apparently of a reflex nature, is at times a prominent symptom. The weight continues to diminish, and without any other symptom the infant may die from exhaustion.

**DIAGNOSIS.**—The diagnosis of infantile atrophy is chiefly to be made from ordinary starvation and from general tuberculosis. From the former it is soon differentiated by its lack of response to good food. In the ordinary cases of starvation which result either from improper food or from lack of food, a diet carefully adapted to the age of the infant or child is soon followed by rapid improvement. The differential diagnosis from general tuberculosis is at times exceedingly difficult. I have had under my care in the hospital in adjoining beds an infant with infantile atrophy



and one with general tuberculosis. In these two cases the symptoms and course of the diseases were so identical that it was impossible to differentiate the two diseases except at the autopsy. On physical examination nothing abnormal could be found in either case except extreme emaciation. In both cases the temperature was slightly raised.

PROGNOSIS.—The prognosis of infantile atrophy is bad, especially during the first year of life. Even under the most careful treatment it is always a very intractable disease. Under special forms of treatment, however, which I shall presently mention, the prognosis is much better than when these cases receive the old and routine treatment of cod-liver oil internally and by inunctions.

TREATMENT.—The treatment of infantile atrophy is essentially by such a modification of the constituents of the milk as to promote intestinal absorption, and without drugs. Although, as I have already stated, it is not entirely proved that the morbid condition is that of a lack of absorption, yet my clinical results are most favorable when the disease has been treated on this principle. After experimenting in a large number of cases by modifying the different constituents of the milk in various ways, I have arrived at the following conclusion: a mixture should be given which contains a low percentage of fat, a high percentage of sugar, and a moderate percentage of proteids. The low percentage of fat is given on the supposition that the infant will increase in weight and thrive on a small proportion of fat, provided it is absorbed. I have found that when higher percentages of fat are given the infant continues to lose in weight. The administration of cod-liver oil is not indicated in these cases, for it is only by a precise adjustment of the percentage of the fat in the food to the individual power of absorption that good results can be obtained. The sugar of high percentage and the proteids of normal percentage seem to be digested and absorbed provided they are combined with a low percentage of fat, since by this combination the nutritive properties of the sugar and of the proteids are made use of. The prescription which I usually write in the beginning of the treatment of these cases, where they occur in the first year of life, is the following (Prescription 79):

PREScription 79.

Fat . . . . .	0 45
Sugar . . . . .	6.00
Proteids . . . . .	1.00
Lime water . . . . .	5.00

After the infant has begun to gain in weight I usually increase the percentage of the fat, but for a number of weeks I do not raise this percentage above 1 or 2. When the infant has once begun to gain steadily the power of absorbing fat is rapidly regained, and percentages such as are in this prescription (Prescription 80) can then be given :

## PRESCRIPTION 80.

Fat . . . . .	3.00
Sugar . . . . .	7.00
Proteids . . . . .	2.00
Lime water . . . . .	5.00

The same treatment can be carried out when the disease occurs in children in their second and third years, but in these cases it is usually possible to increase the percentages of the different elements more rapidly, and after two or three weeks to begin with other articles of diet, such as beef juice, broths of various kinds, and finally, with caution, cereals.

These special modifications of the milk do not, of course, suit every individual infant or child, and when the treatment with them is not successful, each of the elements of the milk must be carefully changed and different combinations of these elements tried until the individual idiosyncrasy of absorption in the special case has been discovered.

I have here a case of infantile atrophy of high grade to show you.

This infant (Case 417) is nine months old.

## CASE 417.



Infantile atrophy. Female, 9 months old.

She has been fed on foods of various kinds, all of which have contained a considerable percentage of starch. She is said to have been healthy and plump at birth and during the early months of life while she was nursed. After she was weaned and placed on these starchy foods she began to lose progressively in weight, and she is now, as you see, in an extremely emaciated condition. Physical examination shows nothing abnormal. She has four teeth. Her temperature is slightly subnormal, her pulse is regular but weak, her respirations are normal. On first entering the hospital the bowels were constipated and the fecal movements were brown and looked poorly digested. Since being placed on a diet of modified milk the movements have become well digested and of normal color, but the total amount in twenty-four hours is greater than normal. She has been very fretful, and at times vomits, but since her diet has been regulated she is less fretful and is somewhat apathetic. On entering the hospital she weighed 2966 grammes (6½ pounds). She has been in the hospital two weeks, and has gained in that time 1000 grammes. The food which has been found to suit her powers of absorption contains fat 1, sugar 5, proteids 1, lime water 5, and 60 to 120 c.c. (2 to 4 ounces) have been given every two hours.

This is a case in which it is uncertain whether recovery will eventually take place, as the emaciation is so extreme, but the prognosis is rendered somewhat favorable by the fact that she has already gained 1000 grammes.

(Subsequent history.) The infant did not increase progressively in weight, but some-

times lost considerably, and at one time it seemed as though she could not possibly live. After the food had been modified in various ways, she finally began to improve, and when she was able to digest and absorb 150 c.c. (5 ounces) of milk so modified as to contain fat 3.5, sugar 6.5, and proteids 1.5, she improved rapidly, and finally recovered entirely. Her temperature, with few exceptions, was normal or subnormal through the whole course of the attack.

Infantile atrophy is so exceedingly intractable a disease, and so greatly taxes the patience and skill of the physician, that it may be of interest and encouragement to you in treating these cases to see this child whom I have had brought to the hospital to show you.

This boy (Case 418) is three years old. He was healthy at birth, and remained strong and well during the early months of his life. He was then fed on a number of starchy

CASE 418.



Recovery from infantile atrophy of high grade. Male, 3 years old.

foods, and soon began to lose progressively in weight. He was in the hospital for five or six months, and was a typical case of a very high grade of infantile atrophy such as I have just shown you. It seemed at one time as though he could scarcely live from day to day, but finally the proportions of the food were so adjusted that he began to absorb a small amount of nutriment. He then began to gain in weight, and recovered entirely. To-day, in his third year, he is, as you see, a remarkably strong, well-developed, and robust child, and, so far as I can detect, is in a perfectly normal condition.

This next infant (Case 419) is also a case of infantile atrophy of high grade.



This infant entered the hospital one week ago, with a history of having been fed on various foods containing starch from the earliest months of its life. It is said to have been healthy at birth and of average weight. On entering the hospital it weighed 2593 grammes ( $5\frac{3}{4}$  pounds). It is, as you see, extremely emaciated, and illustrates the more advanced stage of infantile atrophy. It is unable to raise its head; it is apathetic; its skin is cool and dry; its respirations are shallow; its pulse is weak, and its temperature is slightly subnormal. It

## CASE 419.

## I.



Infantile atrophy. Female, 10 months old.

looks as though it could not live many days. A physical examination shows nothing abnormal in any of the organs. The fæcal movements are rather large in amount, and, since its food has been carefully regulated, are fairly well digested. On entering the hospital they were still larger in amount and were of a brownish color. It weighs to-day 2570 grammes (about  $5\frac{1}{2}$  pounds), which is slightly less than its weight on entering the hospital. This is a case in which the prognosis is very grave, and unless we can soon adjust the food to the digestive tract so as to have it absorbed the infant will die in a short time. It is being fed on a modified milk in which the percentage of fat is 2, sugar 6, proteids 1, lime water 10. Although the skin is cool, it is not so cold as in this next child (Case 420) whom I am about to show you.

(Subsequent history.) In another week the infant began to gain in weight and evidently to absorb its food. Although it had a number of relapses, in which it lost considerably in weight, it finally began to gain steadily. At the end of three months it had recovered entirely, and, as is seen in this picture (Case 419, II.), was quite plump.

## CASE 419.

## II.



Infantile atrophy. Recovery after three months.

In this case the percentage of the fat was finally raised to 4, and that of the sugar to 7, but the proteids had to be kept at 1: the lime water was reduced to 5.

This infant (Case 420, page 874), a female, one and a half years old, entered the hos-

pital two weeks ago. She then weighed 4281 grammes ( $9\frac{1}{2}$  pounds). She is said to have weighed but 900 grammes (2 pounds) at birth. She was nursed by her mother, who apparently had plenty of good breast-milk, and who had two other children whom she had

## CASE 420.

## I.



Infantile atrophy. Female,  $1\frac{1}{2}$  years old.

nursed that were healthy and strong. As the infant did not gain, she was nursed for only a short time, and was then fed on various artificial foods. She began to lose in weight, and this loss has continued ever since, so that now, as you see, her emaciation is extreme.

On physical examination I find that the anterior fontanelle is widely open. There is no

## CASE 420.

## II.



Infantile atrophy, showing extreme emaciation of arms, back, and hips.

enlargement of the epiphyses of the ankles or wrists, but there is a slight rhachitic rosary. Nothing abnormal can be detected in any of the organs. She has four upper and two lower incisors. She is very apathetic, and seems hungry, but when food is given to her she

vomits. Since entering the hospital she has lost 519 grammes ( $1\frac{1}{4}$  pounds). Her skin is dry, harsh, and at times quite cold. It has seemed to me ever since she entered the hospital that there was no hope of saving her life, and, as she is losing in weight and does not respond to the various modifications of the food which have been given to her, the probability is that she will soon die. The faecal movements in this case are very large in amount, but since entering the hospital have been fairly digested. When she is lifted and placed so that you can see her back (II.) you will appreciate the atrophic condition of her muscles, the bones seemingly being covered only by skin. The cervical and inguinal glands are slightly enlarged, and she has a slight cough.

(Subsequent history.) The infant lost steadily in weight during the following week, when it died.

The post-mortem examination, made by Dr. Councilman, showed the following conditions:

There was extreme atrophy of all the muscles. There were no changes in the mesenteric glands, and they were not enlarged, although the extreme atrophy of the mesentery around them made them look so. The liver was normal, and its tissues showed little evidence of atrophy. The spleen was normal. Sections made from various places in the stomach and the intestine showed no changes beyond considerable atrophy of the mucous membrane and of the submucous tissue. The thymus gland was atrophied. There was an extensive bronchitis in the posterior portions of the lungs, while in some parts there was a partial and in others a complete atelectasis.

**ELIMINATIVE.**—Under the term eliminative disturbances of the intestine are included a number of unexplained and obscure symptoms which we at present are unable to classify elsewhere. It is probable that they will be more fully understood in the future. It seems as though the intestine often acts as an organ for the elimination of various morbid products from the economy. The diarrhoea which results from the irritation of these foreign elements is not distinguishable from that which occurs when the irritation is primarily in the intestine itself. Our knowledge of this class of disturbances is, however, so small that I shall merely refer to its possible occurrence.

**ORGANIC.**—The organic diseases of the intestine may be divided into *non-inflammatory* and *inflammatory*.

**NON-INFLAMMATORY.**—The non-inflammatory diseases of the intestine may be divided into *mechanical*, *fermental*, *cholera infantum*, *cholera Asiatica*, and *new growths*.

**Mechanical.**—The mechanical diseases of the intestine are quite numerous, but, with a few exceptions, are not of especial importance medically, and belong rather to the province of surgery.

*Dilatation of the Colon.*—I have already spoken of dilatation of the colon so far as it relates to the diagnosis of dilatation of the stomach. In comparison with dilatation of the stomach, dilatation of the colon is very rare, except as a temporary condition which is liable to occur at any time from an over-production of gas.

I have here an illustration (Case 421, page 876) of dilatation of the colon which was seemingly caused by a congenital stricture, and in which an artificial anus was made by Dr. Halstead. The child recovered from the operation, but later, owing to still further obstruction, he had to be operated upon again, and died.

I show you this case so that if you happen to meet with this rare pathological condition you will recognize its presence. The extreme distention of the abdomen, which



was tympanitic through its whole extent, the evident obstruction to the faecal discharges, and the absence of symptoms pointing towards gastric disease, would suggest a dilatation of some part of the intestine, presumably of the colon.

CASE 421.



Dilatation of colon. Male, 12 years old.

*Volvulus.*—By volvulus is meant a twisting or bending of the intestine. This condition is more apt to occur in early life than later, possibly because of the greater proportionate length of the mesentery at this time, which allows the intestine greater latitude of motion. It occurs either by itself or in connection with the next disease of which I shall speak, from which it is to be differentiated by the absence of blood and mucus in the discharges.

*Intussusception.*—Intussusception or invagination is a condition in which a part of the intestine has passed down into another part. Under these circumstances there is an outer layer of intestine within which is the part of the intestine forming the invagination. Only a small portion of the intestine may be invaginated, or it may extend from the ilco-cæcal valve to the rectum. Small invaginations are frequently found at the post-mortem

examinations of infants and young children. These probably take place during the death-struggle, as no pathological condition is found in connection with them. This form is usually multiple and in the small intestine. The form of intussusception which occurs during life is very rare under three months, and is most common from the third to the sixth month. At this age the large intestine is shorter in relation to the small intestine than in the adult, while the mesentery is relatively wider, and thus allows much greater latitude for misplacement, especially of the cæcum and colon. The etiology of intussusception is obscure, but it is probably directly due to increased local peristalsis.

The pathological condition depends upon the tightness of the constriction and the length of time from the beginning of the obstruction. In some cases the incarcerated portion of the intestine is so little constricted that the bowel remains pervious. In other cases the constriction is so great that the tension of the intestinal capillaries quickly becomes so extreme that hemorrhage occurs, and inflammation, with resulting adhesions, is apt to follow rapidly. The intestine may not only be invaginated, but may be bent on itself, an important point to remember in regard to treatment.

**SYMPTOMS.**—The symptoms of intussusception are usually more acute in infants than in older children. In infants they are often at first rather obscure. Paroxysmal pain and discharges of blood from the rectum occur. Later the blood is mixed with mucus and looks like currant jelly. There is usually vomiting, which may be stereoraceous. The mind is clear, and in young infants the face is often tranquil between the paroxysms of pain, so that on looking at the infant it would scarcely be supposed that a serious condition was present. Later, however, the face grows haggard and the eyes become sunken. During the first twenty-four to forty-eight hours, and even longer, the infants will often take their food quite readily. Tenesmus is at times present. There may be fever, especially when inflammation has occurred. The pulse is usually quickened. These symptoms all vary, and depend on the amount of the invagination. In some cases these are the only signs which indicate that there is abdominal disturbance. In many instances, however, either at once or within a few hours, a tumor can be felt in the abdomen.

**DIAGNOSIS.**—The chief points in diagnosing intussusception are the occurrence of discharges of blood, vomiting, abdominal pain, and the detection of an abdominal tumor, usually on the left side of the abdomen. In these cases a careful rectal examination should always be made, for a tumor can often be found in this way where an external examination has failed to detect it.

**PROGNOSIS.**—Without treatment the prognosis is unfavorable, though there are a certain number of recoveries by spontaneous reduction, or rarely by sloughing of the invaginated portion of the intestine, which is then passed by the rectum. If death takes place, it usually occurs about the third or fourth day, or at any rate within a week, after the incarceration is

complete. Where the incarceration is not complete the infant may live for many weeks, and in older children in rare instances the disease may become chronic.

**TREATMENT.**—The treatment of intussusception when the diagnosis has been definitively made should be immediate, as in no other disease does a delay result in more serious consequences. Food and cathartics or laxatives are contra-indicated. If the infant shows signs of collapse, small quantities of brandy-and-water should be given. In the early hours of the attack an attempt should be made to reduce the intussusception by hydrostatic pressure. This can be easily done by having the infant's buttocks somewhat raised and introducing water under a pressure of about 200 cm. ( $6\frac{2}{3}$  feet) by means of a fountain syringe. The water should be lukewarm, and should have dissolved in it salt in the proportion of one teaspoonful to a quart. The abdomen should be gently rubbed at the same time. In some cases this procedure results in a reduction of the intussusception.

Even where inflammation has not begun and adhesions have not formed, the pressure of the column of water may fail to reduce the intussusception, because the invaginated portion may be bent on itself, so that the hydrostatic pressure increases the obstruction rather than relieves it. Where adhesions have taken place and where there is great congestion, as sometimes occurs during the first twenty-four hours of the attack, hydrostatic pressure is usually unsuccessful and may be dangerous. If this method has failed, the infant should be placed at once in the hands of a surgeon, as under these circumstances an early laparotomy will give the most favorable results.

I shall report to you one of the cases of intussusception which have come under my care.

A male infant (Case 422), six months old, nursed by its mother, and previously perfectly healthy, after a slight loss of appetite for several days began to have abdominal pain in the morning, and in the middle of the day had a discharge of blood from the rectum unmixed with fecal matter or mucus. The bowels had been thoroughly moved on the previous day, and there had been no tendency to constipation. During the afternoon there were five or six discharges of blood. In the evening the infant looked well and did not show any signs of discomfort except occasional slight attacks of abdominal pain and an indisposition to nurse. The rectal temperature was  $39^{\circ}$  C. ( $102.2^{\circ}$  F.). An examination of the abdomen externally and by the rectum revealed nothing abnormal. The infant had a restless night, vomited several times after nursing, and had six discharges of blood. The temperature was  $38.3^{\circ}$  C. ( $101^{\circ}$  F.), the pulse 135, strong and regular, and the general appearance good. The abdomen was soft and not tender on pressure, but towards the umbilicus, under the left costal border, a rather ill-defined cylindrical tumor could be detected.

Hydrostatic pressure was employed to reduce the intussusception, but failed. The surgeon who saw the infant on the second day decided to wait twenty-four hours before performing laparotomy. On the following day the infant died suddenly.

At the post-mortem examination nothing abnormal was found except an ileo-cæcal intussusception. An examination of the invagination showed that the retained cæcum was so twisted that the lower opening was directed to one side of the axis of the intestine, and the hydrostatic pressure from below must have simply packed the sac tighter and rendered reposition more difficult. The invagination involved 20 cm. (8 inches) of the intestine.



The serous surfaces were firmly adherent through their whole extent, and considerable force was required to reduce the invagination without tearing it. The reduction, however, was successfully accomplished, the adhesions giving way and the intestine being left uninjured and apparently healthy. This case illustrates how necessary it is to employ the most skilled surgical aid in these cases.

*Hernia.*—I have already spoken sufficiently of the pathological condition represented by hernia, in my lecture on diseases of the new-born (page 430).

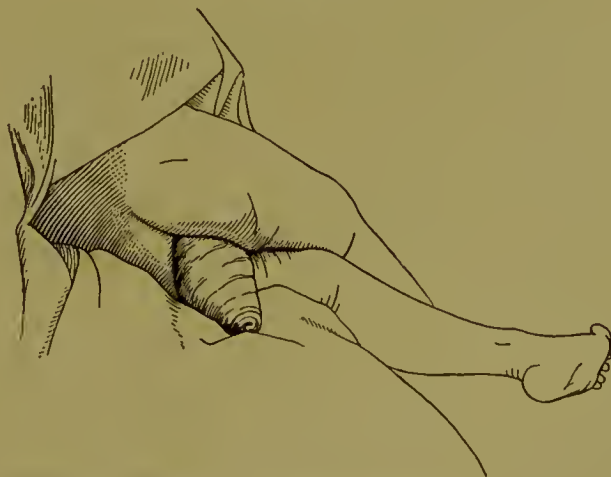
There are a number of lesions which occur about the anus in infants and young children which, though somewhat rare, should be recognized for purposes of differential diagnosis. They are, however, so purely surgical in their treatment that they need only be mentioned here.

*Fissures.*—One of these conditions is that of fissure, which occurs either at the anus or more commonly a little distance from the orifice. Pruritus and reflex urinary symptoms are common. Defecation is often painful, and constipation of the spasmodic type may thus result.

*Prolapse.*—Prolapse of the rectum is not uncommon in young children. It is usually produced by straining from various causes, especially in extreme constipation. The wall of the rectum comes down through the anus, and is easily recognized by the appearance of the mucous membrane. The prolapse is ordinarily transitory, but in the more severe forms the rectum remains down.

The treatment is to remove the cause. Constipation should be relieved first by enemata and then by keeping the movements of the bowels semi-liquid by means of gentle laxatives. The child should be kept in bed for a number of days, the protrusion being gently pushed back each time that it comes down. After reposition it should be kept in place by means of a pad and a T bandage. Under this treatment a large number of cases recover. The more serious and intractable cases, however, should be referred to a surgeon.

CASE 423.



Congenital prolapse of rectum. Female, 22 months old.

I have here a case of prolapse of the rectum to show you which has been under the care of Dr. C. B. Porter.

The infant (Case 423), a female, twenty-two months old, has had this condition of prolapse since birth. Lately the prolapsus has been increasing in size. The infant is not fretful, and seems very well. The movements of the bowels are normal through the prolapsed portion of the rectum. This is one of the more severe types of the disease. You see that the prolapsus forms a large rounded tumor covered with reddish mucous membrane projecting from the anus. It is about 7.8 cm. (3 inches) long and 4.0 cm. (1½ inches) thick. The tumor is not sensitive to the touch.

*Polypi.*—Polypus of the rectum is more common in early life than at any other period. Hemorrhage from the rectum, when not due to constipation, diarrhœa, or fissure, usually arises from polypi. A careful examination for this growth should be made where rectal bleeding is frequent or large. Rectal polypi are of various sizes, and may be myxo-fibromata or adenomata. The surface of the polypus is usually smooth, and the pedicle is often long and thin.

The diagnosis is easily made by a digital examination.

The treatment is simply to twist or cut off the polypi. The growth is not apt to recur.

*Hemorrhoids.*—Hemorrhoids are rarely met with in infancy or early childhood, but can occur as in later life, and should be treated by the same methods.

*Fistulæ.*—Fistula in ano is not a very common condition in infancy or early childhood, but is at times met with. The condition has the same characteristics as in the adult, and should be treated in the same way.

*Fermental.*—The non-inflammatory conditions of the intestine, which for want of a better term we speak of at present as fermental, include those which arise from acid fermentation and albuminous decomposition, which are produced by micro-organisms. The disturbances which arise from these causes represent the greater proportion of the diarrhœal diseases which occur during the warm months of the year.

*ETIOLOGY AND PATHOLOGY.*—The causes of fermental disturbance in the intestine lie in impure or improper foods and bad hygienic surroundings. In both acid fermentation and albuminous decomposition it is probable that the small intestine is most affected. The condition of the mucous membrane may be normal, or there may be desquamative catarrh. The process may go no farther, or it may be followed by inflammatory changes in the intestinal mucous membrane.

The fermental class of cases holds a position midway between the nervous forms of intestinal disturbance and the inflammatory forms with their pronounced lesions.

*SYMPTOMS.*—You can well understand from the great variety of causes which give rise to these fermental processes how varied may be the symptoms. The onset may be subacute, with little or no fever and without vomiting, or it may be acute and accompanied by a high temperature and active vomiting. After a variable period of general discomfort and restlessness, diarrhœa sets in, which varies so greatly as to its frequency, amount, color, and consistency that it would be impossible in the present state of



our knowledge to divide these variations clinically. The onset of fermental diarrhœa is, however, so often characterized by the toxic symptoms of sudden rise of temperature, followed after a day or so by a normal temperature, that when we meet with this occurrence we are usually justified in eliminating the inflammatory and more serious intestinal lesions. In some cases the diarrhœa, although accompanied by much prostration and various nervous disturbances, disappears after a few days; in others, especially in the warm weather, it may last for a number of months. In this fermental diarrhœa the color of the discharges is commonly some shade of green or greenish yellow, and the odor is often very offensive, sometimes being the excessively sour one which is supposed to arise from acid fermentation, and at other times the extremely foul one of albuminous decomposition. The discharges are usually accompanied by considerable pain and a large amount of gas. The symptoms are often so severe that the disease has a serious aspect, but in a considerable number of cases after the intestine has been thoroughly emptied the temperature falls and the nervous symptoms subside. There is usually rapid and great loss of weight. In cases which are not prolonged by fresh irritation or by unwise treatment recovery often takes place quite rapidly.

DIAGNOSIS.—Where the attack is subacute, with slow onset, without vomiting, and with infrequent discharges, the diagnosis is not difficult, and is to be made from the nervous disturbances, which can usually soon be differentiated by the absence of fever and by rapid recovery. Where, however, the onset is acute and is accompanied by vomiting, the diagnosis must often be held in abeyance, as the symptoms of high temperature, vomiting, and diarrhœa may be present in infants and young children in the initial stage of a number of acute diseases. The disease from which it is to be especially differentiated is cholera infantum. In fermental diarrhœa the prostration is much less, and the temperature after the early hours of the attack is much lower. The serous discharges and the continuous vomiting which soon arise in cholera infantum are quite different from the greenish discharges and the less frequent vomiting which occur in fermental diarrhœa. We must remember, however, that cholera infantum and the acute inflammatory intestinal diseases are usually preceded for a number of days by this fermental form of diarrhœa, and that the special micro-organisms which produce the former disease gain an entrance for themselves and their toxins by means of the abnormal intestinal conditions produced by the fermental changes. You must also remember that gastro-enteric symptoms are often so pronounced during the early days of a pneumouia that they may mask the presence of that disease.

PROGNOSIS.—In previously healthy children the prognosis of fermental diarrhœa is good. It depends, however, upon the degree and kind of the fermental process which is causing the disease, and also on the amount of resistance to these processes which the individual possesses. It also depends upon the vulnerability of the individual to the other bacteria which may at



any time complicate the disease. The cases of infantile atrophy which I have just described to you are especially liable to die when attacked by this as well as by any other form of intestinal disturbance. In these cases it seems as though the infant were totally unable to resist even a slight amount of toxic absorption. The prognosis, therefore, when an already debilitated child, or one with infantile atrophy, is attacked by fermental diarrhœa must always be guarded. It also depends upon how soon and in what way the disease is treated.

TREATMENT.—The treatment of fermental diarrhœa is to remove at once the source of the disturbance by thoroughly emptying the intestine. Where the vomiting is excessive it is sometimes necessary to wash out the stomach, but, as a rule, this procedure is not indicated. A dose of castor oil, one teaspoonful for infants under one year, and two teaspoonfuls for older children, is the best initial treatment. In the more severe cases, and where there is a tendency to a prolongation of the acute symptoms, irrigation of the intestine is indicated. Food should be withheld for a number of hours,—at least half a day, if possible. Stimulants are indicated where there is much prostration. Where the stomach is so sensitive that it does not seem advisable to give castor oil, 0.06 to 0.12 gramme (1 or 2 grains) of calomel can be given. The only other drug which in my experience seems to be indicated is bismuth, which should be given in large doses until the disease has run its course and the diarrhœa has ceased.

I have found, contrary to what has been so often stated, that milk can be given after the first twelve to twenty-four hours if it is properly modified. It should contain from ten to fifteen per cent. of lime water, and at first should have the percentages of its elements considerably reduced. The milk which is used for this purpose must be fresh, since it is not sufficient to sterilize it, as the toxic products of bacteria may still be present in it and thus add fresh irritation to that which has already been produced by the fermentation. In many cases it is impossible in the present state of our knowledge to determine what special form of fermentation is present. Where acid fermentation appears to be prominent, the milk should be so modified as to contain a low percentage of sugar and fat, while where albuminous decomposition with its excessively foul odor is met with, the proteids should be reduced to a fraction. Whether this treatment will in the future be proved to be the best it is impossible to state, but on the ground that various forms of bacteria are the cause of these disturbances, and that the special form of bacteria which is producing them has been developed in the food on which it thrives best, it certainly seems reasonable, and should be adopted until further light is thrown upon the subject.

Where breast-milk or fresh modified cow's milk cannot be obtained, weak animal broths, such as those made from mutton, chicken, or beef, can be used. It may perhaps be well to warn you that opium is almost invariably contra-indicated in these cases, and that serious results may arise from

its administration. The peristalsis which occurs as the result of fermental irritation is a conservative process of nature, intended to carry away the morbid products which have resulted from the fermentation. Under these conditions the administration of opium prevents the elimination of the poison from the intestine and allows it to remain and produce still further irritation, or to be absorbed and give rise to still graver septic symptoms. In certain cases where the intestine has been thoroughly emptied, small doses of opium in the form of *tinctura opii camphorata* may be used with caution to diminish pain and control the excessive peristalsis which may result from nervous exhaustion after the disease has run its course. In these cases, however, stimulants are more valuable than opium.

When a child in the warm weather has once had an attack of fermental diarrhœa, it is very apt to have a number of attacks: its diet should therefore be carefully regulated for a considerable period, and, if possible, it should be taken to the sea-shore or the country until the return of cool weather.

As especial illustrations of the great variety of fermental diarrhœas which you are liable to meet with in warm weather, I shall call your attention to these cases which have come under my notice.

A child (Case 424), three years old, and perfectly well, was attacked suddenly with abdominal pain, nausea, pallor, and prostration. He vomited once or twice, and was found to have a temperature of 40° C. (104° F.). Within a few hours he began to have frequent fœcal dejections of sour odor, lessened consistency, moderate amount, and a peculiar dark green color, a specimen of which (Plate III., 18, facing page 112) I have here to show you. This green is one of the more common colors met with in fermental diarrhœa. At first the discharges took place every hour, and later every three or four hours. After the first twenty-four hours the temperature became normal, and in three or four days the diarrhœa ceased entirely.

I have here a case which is also illustrative of this form of fermental diarrhœa.

This infant (Case 425, page 884) is thirteen months old. On entering the hospital it was much emaciated, and had a slight diarrhœa, caused apparently by improper food. Its temperature was only slightly raised. On examining it nothing else abnormal was detected. The diarrhœa was infrequent, and was not accompanied by any other especial symptoms. It soon began to improve, gained in weight, and had a normal temperature. After it had been in the hospital one week it suddenly began to have diarrhœa characterized by large frequent discharges, of lessened consistency, of foul odor, and of the color which you see in this specimen (Plate III., 19, facing page 112). The discharge would seem from its foul odor to be an illustration of what is called albuminous decomposition. You will notice the mixture of yellow and light and dark green, which is so different from the dark-green specimen which I have just shown you (Plate III., 18, facing page 112). These colors are, however, only relative, and are not diagnostic. In this acute attack the temperature was raised at first, but soon fell to a little above normal.

The infant has lost greatly in weight, has become extremely emaciated, and looks as if it would die. The skin often becomes cold, and the prostration is extreme. These symptoms have continued for three or four days, and the number of discharges in the twenty-four hours varies from seven to ten. This is the seventh day from the beginning of the acute attack, and you see the condition in which it has left the patient.

(Subsequent history.) The symptoms became less severe, and the diarrhœa abated. A few days later the diarrhœa stopped entirely, and the infant then gained rapidly in

## CASE 425.

I.



Fermental diarrhœa. Male, 13 months old.

weight and strength. This picture (II.) shows the great improvement which occurred in a month.

## CASE 425.

II.



Fermental diarrhœa. One month after recovery.

These cases of fermental diarrhœa at times are prolonged for many weeks or even months, and thus produce a chronic form of diarrhœa. This occurs especially in children who are the subjects of rhachitis, syphilis, and general tuberculosis; also in those with chronic broncho-pneumonia. I have already told you that the continuous administration of improper food may produce this condition; so also may improper exposure from insufficient clothing.



**Cholera Infantum.**—Cholera infantum is an acute gastro-enteric disturbance characterized by intense choleric form symptoms. The term cholera infantum should be exclusively restricted to this class of cases, and should not be used to designate the many acute and serious attacks of vomiting and diarrhœa which are so often designated cholera infantum. It is a rare disease in comparison with the fermental diarrhœas which I have just described to you.

**ETIOLOGY.**—There is not much doubt that cholera infantum is caused by a specific micro-organism, although this organism has not as yet been determined. It most commonly occurs in the first two years of life, and in its development is probably closely associated with the food, for it has been noticed that infants who are fed exclusively on pure and sterile foods, such as breast-milk, are not liable to be attacked by it. It is also significant that the disease occurs only in hot weather.

**PATHOLOGY.**—The pathology of cholera infantum has not yet been satisfactorily determined, but it seems to be a non-inflammatory disturbance of the whole gastro-enteric tract, without any gross lesion beyond a desquamative catarrh, and sometimes hyperæmia, of the mucous membrane.

**SYMPTOMS.**—The onset of cholera infantum may be sudden, but, as a rule, it is preceded by some form of gastro-enteric disturbance, which, by causing an irritation of the mucous membrane, renders the infant vulnerable. When, however, the disease has once gained a foothold, the development of the symptoms is very rapid.

After a variable but generally short period of restlessness and apparent abdominal discomfort, the infant begins to vomit. The vomiting is either accompanied or quickly followed by profuse diarrhœa. After the stomach and intestine have been emptied of the food which may happen to be in them at the time of the onset, the vomitus and the diarrhœal discharges are chiefly serous; and it is this watery consistency of the discharges which especially characterizes the disease. As a rule, the discharges are odorless, and consist of serum mixed with epithelial cells and many bacteria. Although the disease is more likely to attack weak and debilitated infants, yet it often attacks those who are healthy and robust. It may run its course to a fatal issue in from twenty-four to forty-eight hours. The extremities soon become cold, the skin is pallid or even cyanotic, and the face pinched. The abdomen may be a little distended, but is soft, and soon becomes rather retracted. The pulse is rapid and difficult to count. The respirations are somewhat quick and superficial. The temperature of the entire surface of the body is low, but the deep rectal temperature is high,  $39.4^{\circ}$ ,  $40^{\circ}$ , or  $40.5^{\circ}$  C. ( $103^{\circ}$ ,  $104^{\circ}$ , or  $105^{\circ}$  F.). The thirst is great and is a very prominent symptom. The fontanelle very soon becomes depressed. The urine is suppressed, and nervous symptoms, such as twitching of the arms and great restlessness, are present. Rapid emaciation takes place, and all the symptoms increase in severity. At first the infant whimpers, but soon it becomes listless, falls into a stupor, or may have convulsions. The

infant may die in this stage, which closely resembles the algid stage of cholera Asiatica. The disease appears to be self-limited, and if the infant survives the first two or three days a crisis comes, the skin becomes less cool and of a better color, the vomiting and diarrhœa grow less frequent, and finally it is left with a slight amount of simple diarrhœa and occasional vomiting. These symptoms may become chronic, in which case the infant finally dies of exhaustion or from an attack of one of the other gastro-enteric diseases, to which it is left very susceptible.

DIAGNOSIS.—The diagnosis of cholera infantum is not difficult if the characteristic symptoms are borne in mind; these are rapid onset, constant vomiting, frequent serous discharges, intense thirst, high rectal temperature, low surface temperature, collapse, depressed fontanelle, sudden loss of weight, and distressed, restless expression, suggesting speedy death, all developing in from twenty-four to forty-eight hours.

PROGNOSIS.—The prognosis is bad. The more violent the attack, the higher the temperature, the less the vitality, and the warmer the weather, the worse is the prognosis. When the infant has survived the very acute symptoms which appear in the first two or three days, the prognosis is much more favorable.

TREATMENT.—Cholera infantum is so formidable in its attack that it must be treated most energetically if we hope to succeed in saving the infant's life. The indications for treatment are (1) to assist the effort which nature is making to free the stomach and intestine from the poison which is in them; (2) to restore the surface circulation, which is so seriously interfered with; (3) to supply water to the tissues, which are being drained to so grave an extent; and (4) to support the strength until the disease has run its course.

The poison seems to act with especial virulence on those portions of the economy where it is most concentrated,—namely, the stomach and the intestine. We therefore have at first extreme irritation of these parts, which causes increased peristalsis, and later vaso-motor paralysis, with great transudation of serum. This condition of the gastro-enteric tract is to be especially borne in mind during the whole course of our treatment.

In this disease we should not attempt to use any remedy which works slowly. The condition of the mucous membrane is in all probability such that absorption of drugs does not take place readily. The administration of drugs is, therefore, contra-indicated, for they may later, when absorption is being restored, prove fatal by their cumulative action. During the acute stage of the disease the digestive functions fail to act, and therefore food of any kind will be only an additional source of irritation.

Early in the attack, and when the vomiting has not caused much prostration, the stomach should be thoroughly washed out with warm water and the intestine should be irrigated. If the rectal temperature is very high, ice-cold water may be used for irrigation. Where the vomiting has continued for some time and there is prostration with great thirst, the infant



should be allowed to suck sterilized ice-cold water from the bottle. At first nothing else should be given by the mouth.

The infant should be placed at once in a warm pack. This should be done by wrapping it to the chin in sheets wrung out of water at least as hot as 38° C. (100.4° F.). It should then be enveloped in a hot blanket. This procedure should be repeated as often as the infant shows signs of collapse or much cyanosis and coldness of the skin. This is the best method that I know of to restore the surface circulation. In extreme cases the subcutaneous injection of salt solution can be tried.

While the infant is in the hot pack, water can be given freely by the mouth, and, if necessary, small and frequently repeated doses of stimulants, unless they appear to excite vomiting, in which case they should be given hypodermically.

If the vomiting and diarrhoea still continue excessive after this treatment, small doses of morphine, 0.0006 gramme ( $\frac{1}{100}$  grain), and atropine, 0.00008 gramme ( $\frac{1}{800}$  grain), for an infant a year old, can be tried hypodermically. The effect should be carefully watched, and the dose repeated if necessary, as recommended by Holt.

If, after the vomiting and diarrhoea have ceased, the heart's action continues very weak and does not respond to stimulants, small doses of digitalis should be given. The greatest caution should be employed in using drugs, however, as they generally do more harm than good.

If an absolutely fresh and sterile milk can be obtained, it can be used as a food, as in any of the other forms of gastro-enteric diseases which I have already described, but for some days the percentages of the elements in the milk must be much lessened, and the child's strength must be supported mostly by stimulants freely diluted with sterilized water.

**Cholera Asiatica.**—Cholera Asiatica is a highly infectious disease, caused by the comma bacillus of Koch, which manifests its most violent symptoms in the gastro-enteric tract. Its symptoms very closely resemble those of cholera infantum. The disease in infants should be diagnosed from cholera infantum, which is done by finding the comma bacillus in the vomitus or in the discharges. There are no especial differences between cholera Asiatica in the adult and the same disease in the infant. It is exceedingly fatal during infancy and childhood, and young infants who are attacked by the disease during a cholera epidemic seldom live. The treatment is the same as that which I have just described for cholera infantum.

**New Growths.**—New growths in the enteric tract are very rare in infancy and childhood, and are mostly confined to myxomatous polypi of the rectum.

**INFLAMMATORY.**—The inflammatory diseases of the enteric tract may be *acute* or *chronic*.

Under *acute* inflammatory diseases may be included *appendicitis* and *ileo-colitis*.



**Appendicitis.**—Inflammation of the appendix cæci is essentially a surgical disease, and is one which under all circumstances should be placed immediately in the hands of those who are skilled in abdominal surgery. From my observation of this disease I am so strongly impressed with this fact that I consider an extended description of it in medical lectures and by physicians out of place. I shall therefore confine my remarks on this disease to a very few words, which will aid you in making a diagnosis when you meet with one of these cases.

Under the term appendicitis we now include those inflammatory conditions in the neighborhood of the cæcum which were formerly called cæcitis and perityphlitis. The reason for this is that there is little doubt that in most instances the appendix is the part primarily involved. The disease occurs most commonly after the tenth year, and is rare in the early months of life, but it may occur at any age.

**ETIOLOGY.**—The cause of appendicitis is in most cases an inflammation of the lining mucous membrane of the appendix arising from faecal concretions. It is seldom caused by any foreign bodies, such as seeds of any size.

I have here a specimen of the appendix (Fig. 111) which was taken from a girl nine years old forty-eight hours from the beginning of the attack, the first she had ever had. The operation was performed by Dr. S. J. Mixer, and was followed by complete recovery.

FIG. 111.



Appendix removed from female 9 years old. (Natural size.)

On opening the appendix this faecal concretion was found (Fig. 112).

FIG. 112.



Faecal concretion in appendix. (Natural size.)

**PATHOLOGY.**—The pathological lesions which occur in these cases vary from a simple inflammatory condition, with exudation, induration, and thickening, to gangrene and necrosis.

**SYMPTOMS.**—The symptoms of appendicitis are, as a rule, the more obscure the younger the individual. In infants and young children abdominal pain may be difficult to localize, and may be referred to some other part of the body. In like manner pain in the thorax may be referred to the abdomen, so that it is often impossible to be guided by the apparent seat

of the pain. There are no prodromal symptoms which are especially characteristic or of much aid in determining whether appendicitis is present. The temperature is often very misleading. I have seen a child with a severe attack of appendicitis in whom the acute symptoms disappeared in a few hours and the temperature was raised very little above normal. Dr. Mixer, whose surgical knowledge was called upon to determine what should be done in this case, decided to operate, and on opening the abdomen the appendix was found in a highly inflamed condition: pus had formed and distended it, and perforation had almost taken place. There is nothing especially significant in the pulse or the respiration which will aid you in diagnosing the disease. In a number of cases, however, vomiting, pain and tenderness in the region of the cæcum, and later a sensation of resistance and dulness on percussion, constitute a group of symptoms which should lead us strongly to suspect the presence of this disease. The vomiting, as a rule, is not stercoraceous, and in young infants diarrhœa is apt to occur as often as constipation.

In cases of appendicitis which recover after operation various inflammatory lesions are left, and the disease is liable to recur from time to time. This condition is known as chronic appendicitis. Where the symptoms continually recur, the patients lose in weight and strength, but often can be entirely cured by having the appendix removed.

DIAGNOSIS.—The diagnosis is to be made chiefly from intussusception and volvulus, especially the former. In intussusception, as I have already told you, there is usually an absence in the beginning of pain and tenderness, and the tumor which is ordinarily found is to the left of the median line rather than to the right. The vomiting in appendicitis is not stercoraceous; in intussusception it is often so. Pain and tenderness to a varying degree are always present, but the tumor is often not felt until late in the disease. The temperature and pulse are generally slightly raised. The same anxious expression of the face occurs in appendicitis as in intussusception. You must not depend upon the locality of the tumor and the pain and tenderness in differentiating these two diseases, for in some instances the inflamed appendix may be found to the left of the median line, and in intussusception, especially if not of the ileo-cæcal variety, it may be on the right of the median line. All these questions, however, are for the skilled surgeon to decide; and when this group of symptoms is present we are justified in making a provisional diagnosis of appendicitis and in at once summoning surgical aid.

PROGNOSIS.—The prognosis of appendicitis under judicious treatment, especially if operative interference is instituted early, is very favorable; but when operation has been deferred until perforation has taken place the prognosis becomes unfavorable. Even under these conditions, however, many cases recover. The prognosis of cases which are operated upon when inflammation is not present between recurrent attacks of appendicitis is in almost every instance favorable.



**TREATMENT.**—When you have made the diagnosis of appendicitis, you should at once place the child in bed, enforce absolute quiet, apply hot fomentations to the abdomen, and, if necessary, give sufficient opium to relieve the acute pain. Cathartics and laxatives should not be given. The food should be small quantities of peptonized milk, and should be given by enemata. It is almost needless to repeat that the best surgical aid should be called in at once to determine upon the next steps in the treatment.

I have to report to you a case of appendicitis which was under the care of Dr. Crocker and was operated upon by Dr. George Haven.

A child (Case 426), twenty-eight months old, had loss of appetite, sleeplessness, nausea, vomiting, slight diarrhoea, and abdominal pain. Two days before the operation a tumor had been found in the left lower part of the abdomen. The child's face had a pinched expression and showed much pallor. Her pulse was 170, her temperature 39.7° C. (103.5° F.). On the day when the operation was performed, in addition to the tumor which had first been found, the right half of the abdomen was filled by a tumor of somewhat irregular outline, with tense walls, and giving an absolutely flat note on percussion. Changes of posture produced no effect on the physical signs. An incision was made through the middle of the tumor, and about a pint of pus escaped, together with masses of detritus having a strongly faecal odor. The child recovered entirely.

I happen to have here in the wards a little girl (Case 427), eight years old, who illustrates one of the mild cases of appendicitis which often recover without operation, and whom I have placed under surgical supervision in accordance with my strong opinions on this subject.

#### CASE 427.



Appendicitis. Female, 8 years old.

This child was well until four days ago, when she began to have severe pain in the right side in the region of the appendix. This was followed by headache, nausea, and vomiting. Marked tenderness soon appeared in the area where she complained of pain. The pain continued with slight intervals. The bowels were constipated. At first there was an almost constant desire to have a movement of the bowels. On entering the hospital the abdomen was tympanitic and not tender, except in the area which I have marked in black, which covers a space of 6.5 to 7.8 cm. (2½ to 3 inches). Within this line there have been pain, tenderness, and dulness on percussion. Her tongue has been coated. On



close inspection you will notice that there is slight bulging in the area marked in black. The temperature has been about 39.4° C. (103° F.), the pulse 100, the respirations 28.

With symptoms of this nature there is not much doubt that we are dealing with a case of appendicitis. Morphine, 0.004 gramme ( $\frac{1}{16}$  grain), was required a number of times to relieve the pain.

(Subsequent history.) On the day following the child's entrance to the hospital the temperature began to fall, the tumor became less distinct, and there was less tenderness and not much pain. It was decided not to operate, but to watch the case carefully. On the third day after entering the hospital, the seventh day of the disease, the temperature became normal, the pain and tenderness disappeared entirely, and the tumor became indistinct. The bowels moved naturally on the seventh day, and in the third week from the onset of the attack the child was perfectly well, and only a little resistance could be felt in the area which had been occupied by the tumor.

**Ileo-Colitis.**—Under the term ileo-colitis are included all the more marked and grave lesions of the intestine. These lesions are so varied that it would be impossible to classify them in detail, and practically we can divide them in only a very general way.

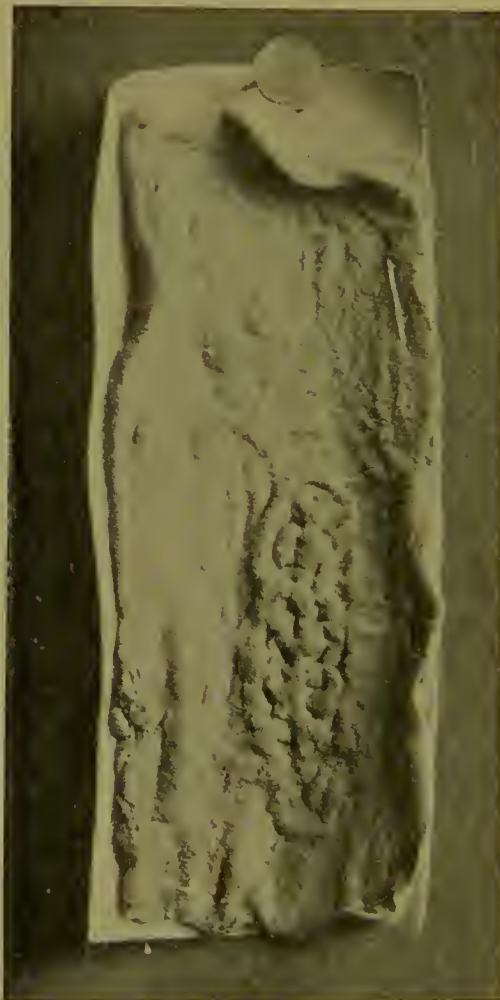
The divisions which have been adopted to simplify the subject are (1) simple catarrhal inflammation, which includes the non-ulcerative form of follicular inflammation, (2) follicular ulceration, (3) an inflammation characterized by a pseudo-membrane, (4) an inflammation caused by the typhoid bacillus, (5) an inflammation caused by the amoeba coli, and (6) an inflammation caused by the bacillus tuberculosis. The first three of these divisions, *catarrhal*, *ulcerative*, and *membranous*, although differing essentially in their prognosis, are so often represented by the same symptoms that they can be differentiated only in the most general way. A symptom common to all these diseases is that the temperature, although not necessarily high, is, as a rule, raised through the whole course of the disease. In this way we can usually differentiate these diseases from the non-inflammatory conditions of which I have already spoken. There are so many varieties of pathological lesions found in connection with the catarrhal and non-ulcerative follicular and the ulcerative follicular inflammations that the clinical distinction between the two conditions, until our knowledge of these diseases shall have been greatly increased, must be very limited. In both the lesions are so varied that they probably arise from a number of organisms, and their pathology must for the present include all forms which cannot be classed under the pseudo-membranous, typhoidal, or amoebic forms of ileo-colitis. They may occur as acute primary diseases, but are usually secondary to the fermental diarrhoeas, and sometimes to the infectious diseases, especially measles.

In the pseudo-membranous form of ileo-colitis the ileum and the colon are chiefly affected. The lesions are probably due to a number of organisms, but its pathology is more definitely known than that of the catarrhal and ulcerative follicular forms. It is characterized by the presence of a membrane on the surface of the mucous membrane, which extends into it, due to a combination of fibrinous exudation and necrosis. That is, there is a definite pathology. The disease may be primary or secondary. In the primary

form it represents what is usually spoken of as epidemic or sporadic dysentery. The secondary form is that which follows certain infectious diseases, such as measles. All these forms are commonly spoken of as dysentery; but from what I have told you you will see that the word dysentery should no longer be retained in our nomenclature, as it has been used for so many different pathological conditions.

Before endeavoring to tell you what little is known regarding the symptoms of these diseases, I shall show you a few specimens illustrating some of the pathological conditions which occur in *ileo-colitis catarrhalis*, *ileo-colitis ulcerativa follicularis*, and *ileo-colitis pseudo-membranosa*. Much more extended studies of these conditions, both as to their pathology and their bacteriology, must be made before anything more than this general view of the subject can be used for clinical purposes. You will of course understand that these specimens which I am about to show you do not represent all the lesions which occur in these diseases, but illustrate some of the principal ones only. The notes in connection with these cases show how with our present knowledge it is usually impossible for us to diagnose the lesions during life.

FIG 113



Hyperplasia of the lymph-follicles. Warren Museum, Harvard University.

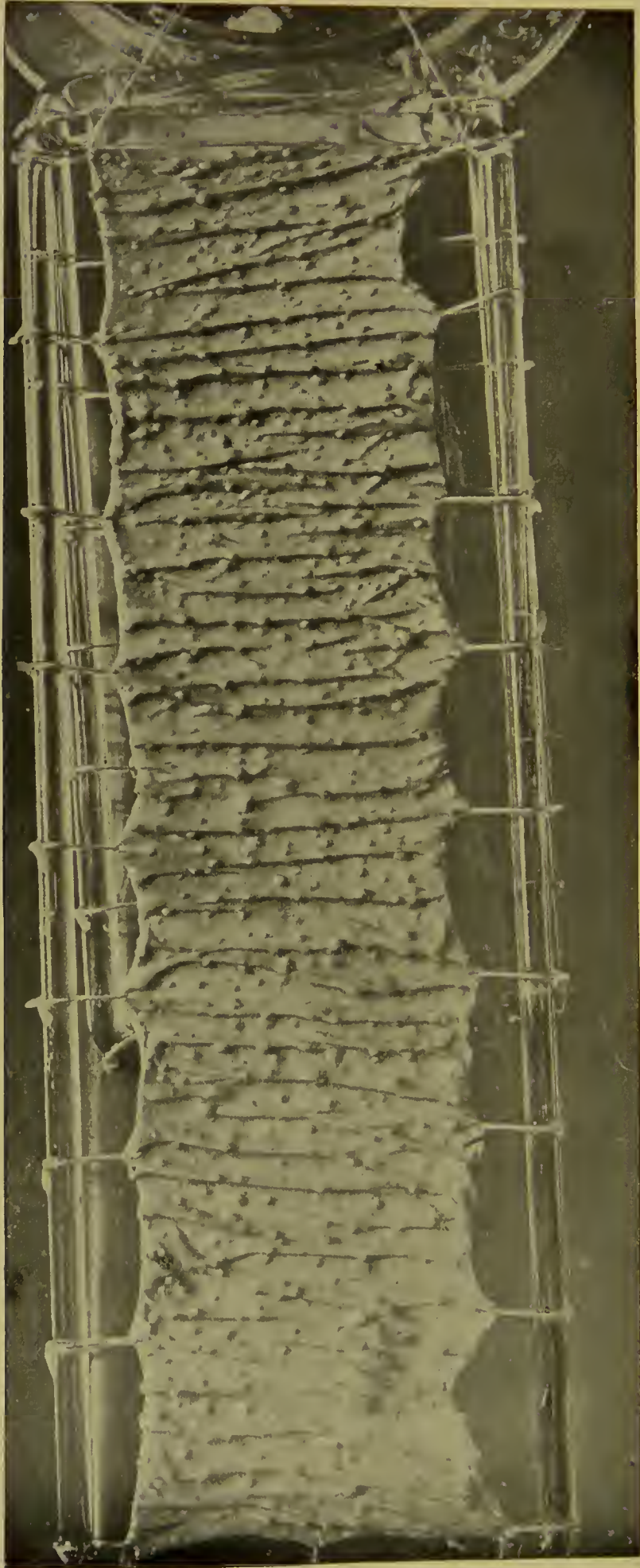
This first specimen (Fig. 113) is a portion of the colon of an infant who during life had only a slight diarrhœa.

CASE 428. FIG. 114.

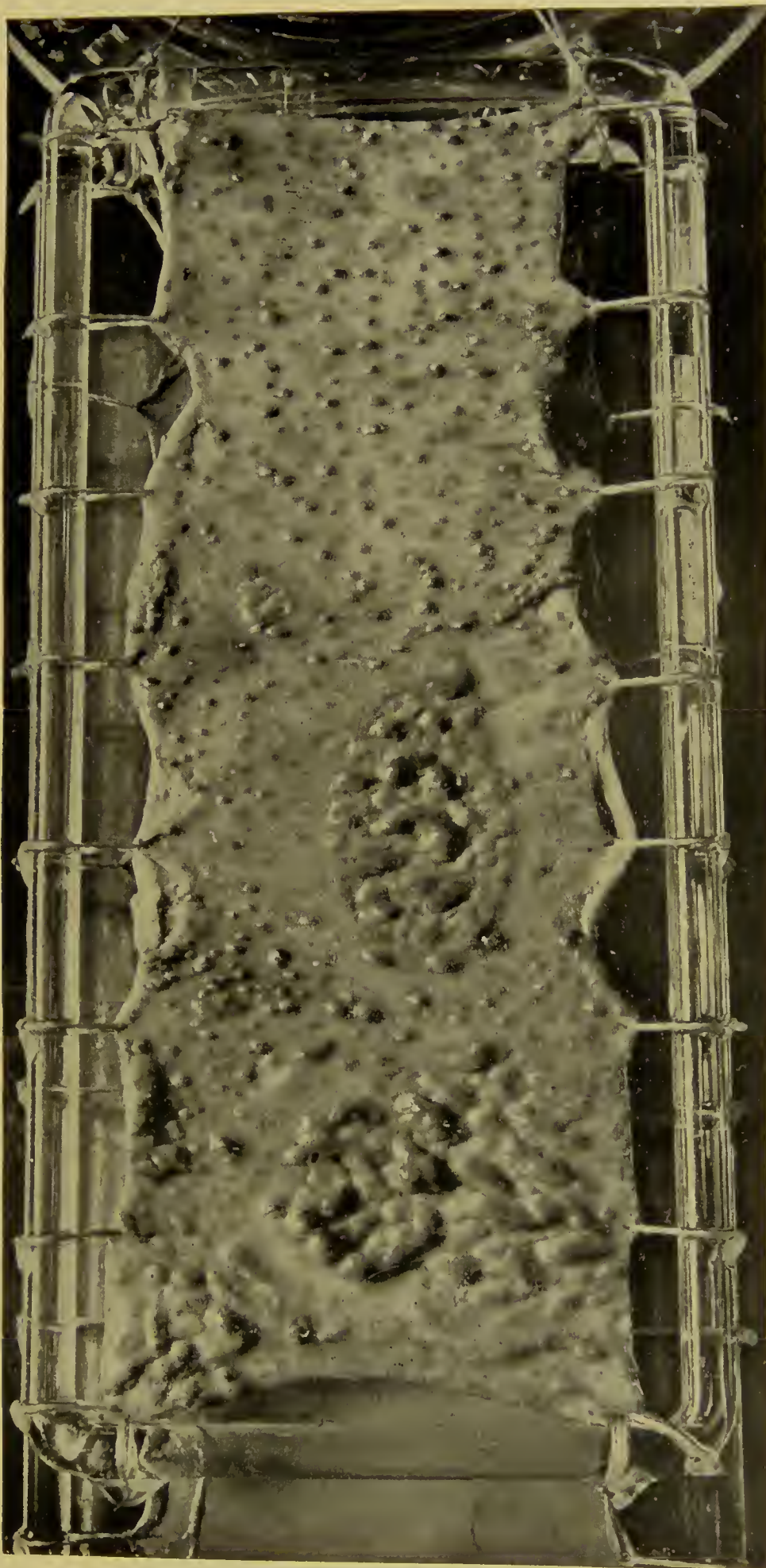


Non-ulcerative follicular inflammation. Simple hyperplasia of lymph-follicles.  
Female, 3 years old. Warren Museum, Harvard University. (Page 893.)





Colitis follicularis non-ulcerativa. Male, 2 years old. Museum of the College of Physicians and Surgeons, New York. (Page 893.)



Colitis follicularis non-ulcerativa. (Page 893.)

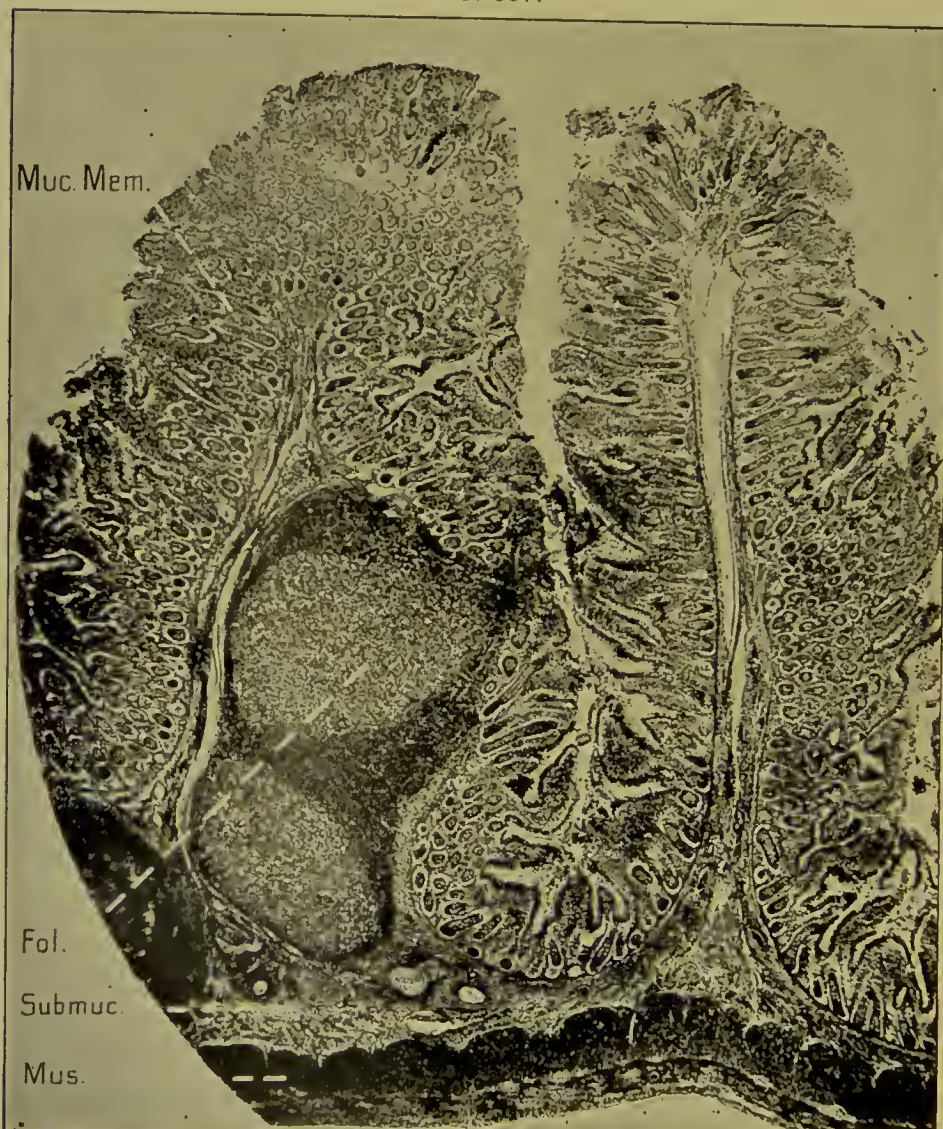


FIG. 116



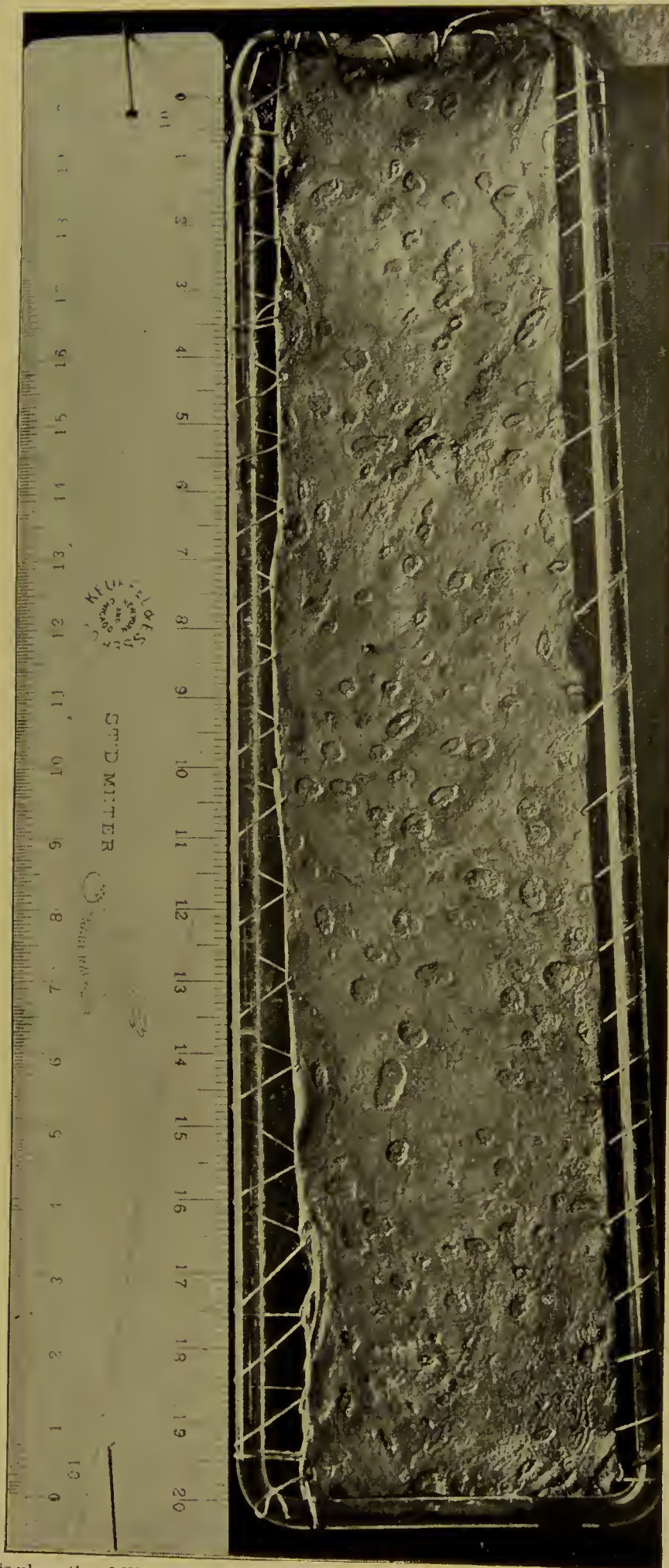
Hyperplasia of lymph-follicles (solitary glands). Muc. Mem., mucous membrane ; Lym. Ts., lymph-tissues ; Mus., muscle ; Fol., follicles. (Page 893.)

FIG. 117.

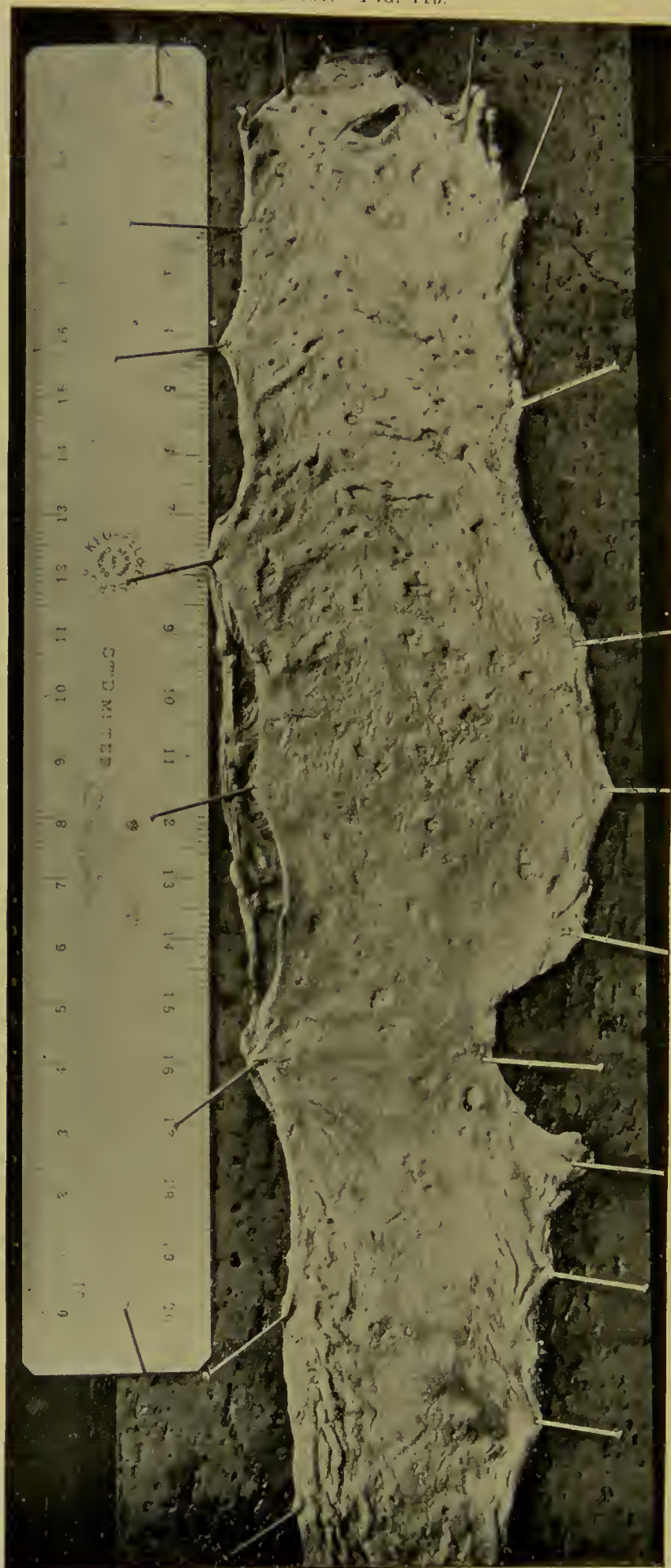


Muc. Mem., mucous membrane ; Fol., follicles ; Submuc., submucous tissue ; Mus., muscle. (Page 893.)



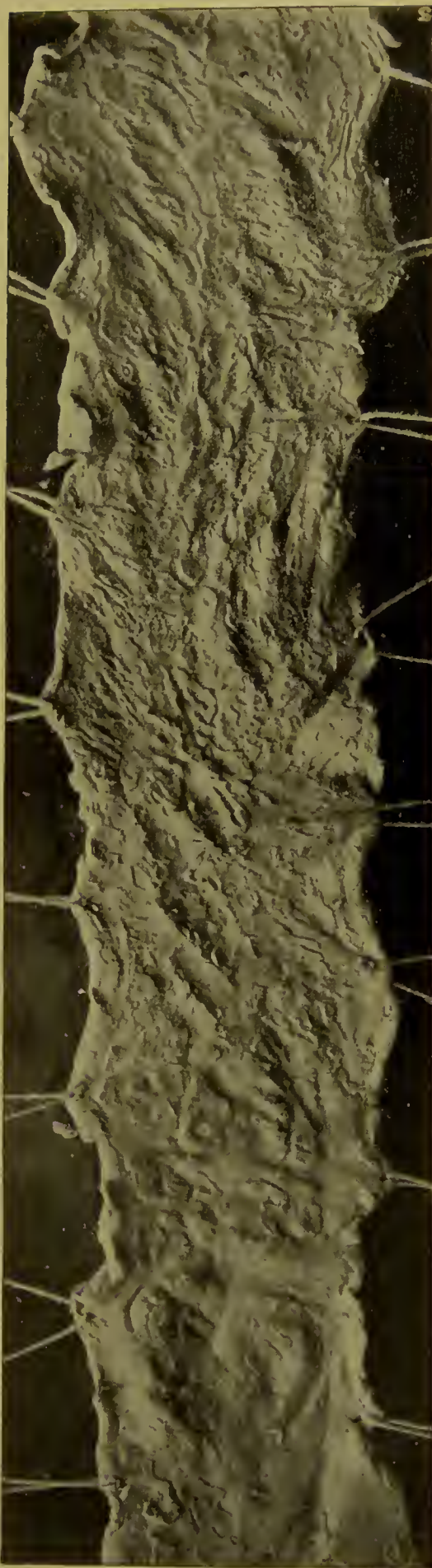


Ileo-colitis ulcerativa follicularis. Infant, 16 months old. Museum of the College of Physicians and Surgeons, New York. (Page 893.)



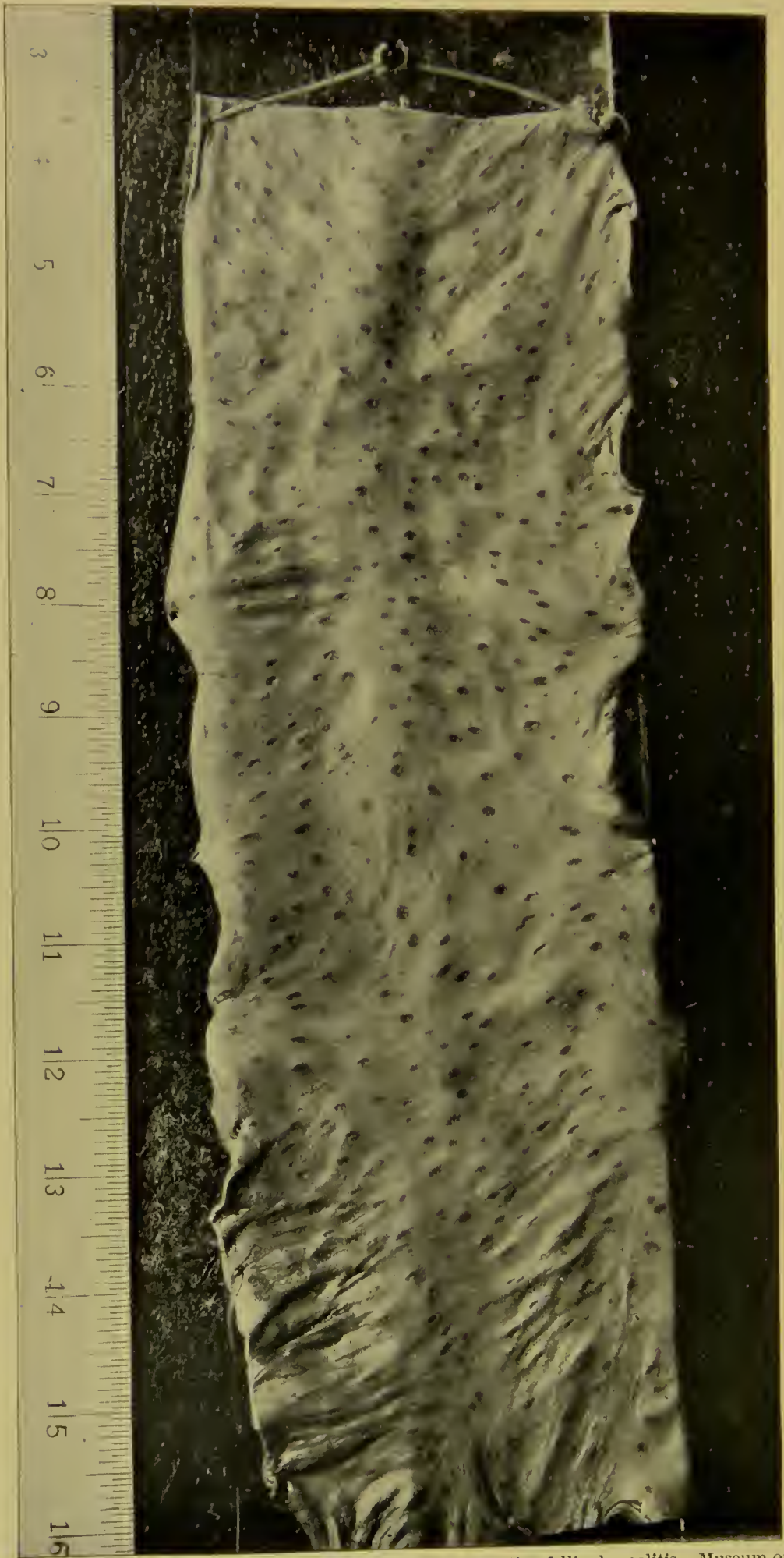
Acute ulcerative catarrhal colitis. Female, 3 months old. Museum of the College of Physicians and Surgeons, New York. (Page 894.)





Inflammation of follicles and surrounding parts of colon. The process has gone on to necrosis. Female, 3 months old. Warren Museum, Harvard University. (Page 894.)





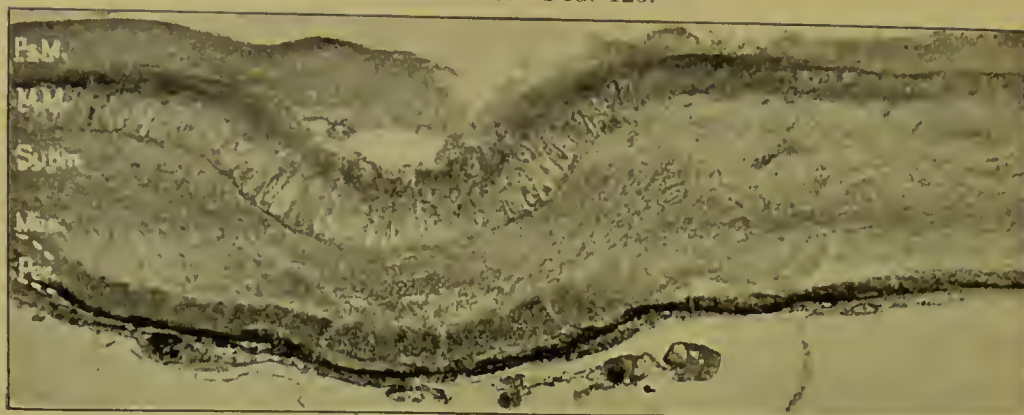
Pigmented follicular ulcers of colon. Chronic catarrhal ulcerative follicular colitis. Museum of the College of Physicians and Surgeons, New York. (Page 894.)



Pseudo-membranous colitis. Child,  $3\frac{1}{2}$  years old. Museum of the College of Physicians and Surgeons, New York. (Page 895.)

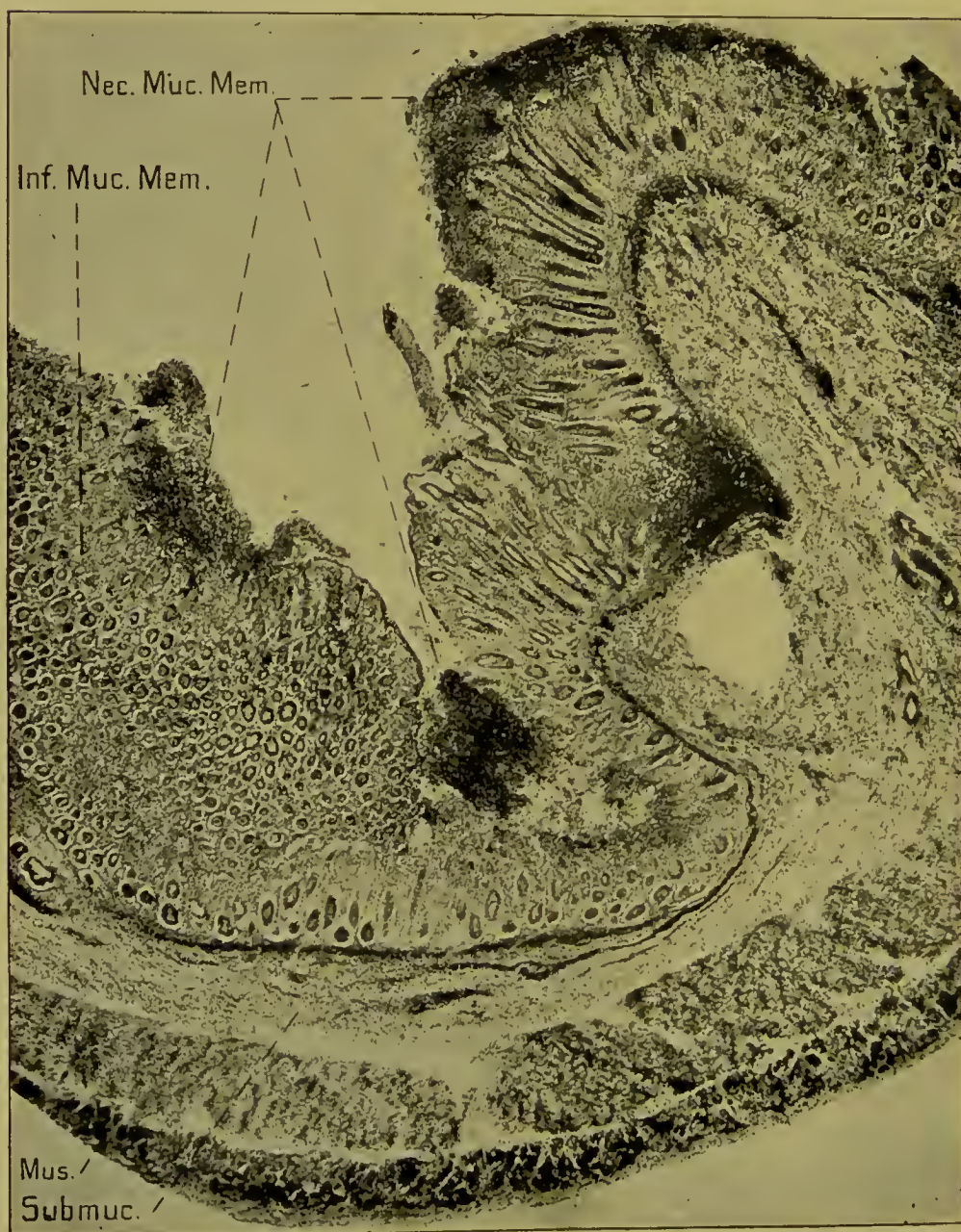


CASE 435. FIG. 123.



Pseudo-membranous colitis. Female, 4 years old. Ps. M., pseudo-membrane ; M. M., mucous membrane ; Subm., submucosa ; Mus., muscle ; Per., peritoneum. (Page 895.)

CASE 436. FIG. 124.



Nec. Muc. Mem., necrotic mucous membrane ; Inf. Muc. Mem., inflamed mucous membrane ; Mus., muscle ; Submuc., submucosa. (Page 895.)



You see that the lesion is quite marked and simulates closely the hyperplasia of Peyer's patches which is commonly seen in typhoid fever; but in this case it represents merely intestinal irritation.

This next specimen (Fig. 114) was found at the autopsy of a little girl, three years old, who had been under the care of Dr. Webber.

The child (Case 428) was attacked with excessive vomiting after eating pigs' feet, and the vomiting continued until her death, five days later. The lesions are chiefly in the upper part of the colon, and consist of a general non-ulcerative follicular inflammation. The hyperplasia of Peyer's patches is, as you see, extreme.

Through the kindness of Professor W. P. Northrup I am enabled to show you some interesting specimens of lesions of colitis which occurred in his practice, and which are now preserved in the Museum of the College of Physicians and Surgeons, New York.

This specimen (Fig. 115) is one of acute catarrhal follicular inflammation without ulceration.

The infant, a male (Case 429), two years old, entered Professor Northrup's service with a history of diarrhoea and general debility lasting two weeks. While the infant was in the hospital there was a continued high temperature, which at one time reached 40° C. (104° F). The symptoms were mostly of a cerebral type, and the abdominal symptoms were not severe or prominent enough to indicate the marked lesions which were found at the autopsy. The post-mortem examination, made by Professor Northrup, showed the following conditions:

Brain normal.

Stomach congested.

The small intestine contained a large amount of thick mucus. The solitary follicles were enlarged, rather more in the upper third of the intestine. Peyer's patches were markedly swollen, and a few solitary follicles appeared to be ulcerated. The mesenteric lymph-glands were enlarged.

The mucous membrane of the colon was swollen: the follicles were enlarged and somewhat pigmented, but not ulcerated.

Here is another portion of the colon (Fig. 115, II.) taken from the same infant (Case 429).

As you see, the solitary follicles are very much enlarged, and in Peyer's patches, which are in the middle of the specimen, the hyperplasia is of a very high degree.

I have also here some microscopic sections of this form of follicular inflammation. In this first specimen (Fig. 116) you will see the great enlargement of the lymph-follicles.

In this next specimen (Fig. 117) you will notice the inflamed condition of the mucous membrane as well as the enlarged lymph-follicles.

This next specimen (Fig. 118) was taken from an infant (Case 430) sixteen months old.

The infant before entering the hospital had had occasional attacks of diarrhoea for three months, presumably caused by improper feeding. Soon after entering the hospital it rapidly grew worse and died.

The autopsy, made by Dr. Northrup, gave the following results. No tubercular lesions. Bronchial lymph-follicles enlarged. Small intestine showed much swelling and congestion of Peyer's patches, but no ulceration. The colon showed extensive follicular ulcerations. In the small intestine and the colon were found masses and strings of greenish mucus; no blood.

This next specimen (Fig. 119) was taken from a female infant (Case 431), three months old.

The infant on entering the hospital was somewhat rachitic, emaciated, and fretful. There were no vomiting and no fever. It took very little nourishment, and at this time was having one large, watery, faecal discharge daily. The faecal movements were greenish yellow. The infant apparently improved for about a week. The temperature was then found to have risen, and during the next week it varied from 36.6° to 37.7° C. (98° to 100° F.). During the next week the temperature was sometimes subnormal. At the end of three weeks the infant began to fail rapidly without any discoverable cause, and died suddenly.

The autopsy was made by Professor Northrup, and showed the following lesions: the mucous membrane of the ileum was swollen, and the lymph-follicles were enlarged, but not ulcerated.

The report of the examination of the colon, made by Professor Delafield, was as follows. Numerous ulcers, some round and some irregular in shape; an increased production of mucus; a profuse growth of connective tissue between the tubules, with disappearance of the tubules; necrosis of the new tissue so as to form ulcers; the solitary follicles swollen, but not concerned in the formation of ulcers, which are simply necrotic. No amœbæ found. The process is one which would ordinarily come under the head of acute catarrhal colitis.

The next specimen (Fig. 120) is one which I am enabled to show you through the kindness of Professor Holt.

The infant (Case 432) was three months old, and was in the hospital under the care of Dr. Holt. It had no acute symptoms, but had never been well, and before entering the hospital had lost in weight and strength. It entered the hospital for vomiting and diarrhœa. Nothing was found on physical examination. While in the hospital it had from six to eight loose greenish discharges in the twenty-four hours, and vomited occasionally. Its temperature varied from 37.2° to 38.3° C. (99° to 101° F.). It gradually failed, and died twelve days after entering the hospital.

The post-mortem examination, as you see (Fig. 120), shows extensive follicular ulceration of the colon, especially in the lower part of the specimen, where there is a large ulcer. The tissues around the follicles are also involved, and the process has gone on to necrosis.

This next specimen (Fig. 121) was taken from a male infant (Case 433) six months old, also a patient of Dr. Northrup's.

The infant when it entered the hospital was in a very wasted condition, and died in a few days without any especial abdominal symptoms.

The autopsy, made by Dr. Northrup, showed numerous superficial abscesses on the body, a general bronchitis, and a beginning broncho-pneumonia. The lesions in the intestine were an inflammation of the solitary follicles of the ileum and of the colon, with small ulcerations at the apices of the follicles in the colon, no ulcers being present in the ileum. In the specimen these ulcers are, as you see, pigmented, which denotes a chronic condition.

The apices of the follicles are sometimes found pigmented as the result of post-mortem changes, and may simulate these ulcerations.



The next specimen (Fig. 122) is one of pseudo-membranous colitis.

This child (Case 434), three and a half years old, a patient of Dr. Northrup's, entered the hospital in a very reduced condition following an attack of whooping-cough. It was attacked with diphtheria, and during the ten days that it was suffering from this disease there was a slight amount of diarrhœa, but no pain and no tenesmus.

The autopsy showed this pseudo-membranous inflammation through the whole length of the colon, most marked in the lower third. The other organs were normal. The microscopic examination of the colon confirmed the diagnosis of pseudo-membranous colitis.

I have here a microscopic section (Fig. 123) of another case (Case 435) of pseudo-membranous colitis.

This child, a female, four years old, was a patient of Professor Northrup's. It had always been delicate. It had pneumonia twice in its fourth year. Eight days before its death it was attacked with vomiting and diarrhœa. There was blood in the fœcal discharges. The pulse was rapid. The loss of strength and the pallor were marked. The eyes were sunken, and the tongue was dry. On the last day of its life it became very feeble, and died in convulsions. Early in the disease the discharges were frequent. Later, they were from four to six daily, and were accompanied by tenesmus and tenderness of the abdomen.

The autopsy showed that the mesenteric lymph-follicles were not much enlarged; the follicles in the colon were slightly enlarged. The whole intestine was injected in patches, and contained fœcal masses of a yellowish color. The large intestine was filled with large quantities of fœces of foul odor and colored by bismuth. The whole surface was rough, and did not look like a mucous membrane, but rather as though a thin layer of gelatin had been poured over it. This film could be pulled away with the forceps. The solitary follicles were enlarged.

The microscopic section of this specimen shows a marked fibrino-purulent exudation, forming a membrane which characterizes the disease as pseudo-membranous colitis.

I have also here to show you, through the kindness of Professor Northrup, an interesting specimen (Fig. 124) of an intestinal lesion in connection with the pseudo-membranous condition which you have just seen.

This child (Case 436), three and a half years old, had whooping-cough. It was then attacked with diphtheria, and during the course of the disease the temperature was raised continuously, at times being as high as 40° C. (104° F.). During this attack it had diarrhœa with blood in the discharges, but no pain or tenesmus and no other symptoms of colitis.

The autopsy showed a broncho-pneumonia, and a normal condition of the stomach and small intestine. The colon showed an apparent exudation, which simulated that of a pseudo-membranous colitis so closely that before the microscopic examination was made it was supposed to be identical with the pathological lesions found in the case of pseudo-membranous colitis (Case 435) which I have just shown you. The surface appearance in the fresh specimen was identical. Under the microscope, however, the lesion proved to be only a superficial necrosis of the mucosa, with swelling of the lymph-follicles.

This specimen should impress upon you how important it is not to rely upon the macroscopic appearances of intestinal lesions without microscopic corroboration.

Now that you have seen these pathological lesions, you will understand why it is often impossible to differentiate them clinically from one another. I shall, therefore, speak of them together.



As illustrations of the difficulty and in many instances the impossibility of diagnosing intestinal lesions I shall report to you some cases which have been under my care.

One of these cases was that of a little girl (Case 437), five years old, who during the hot weather in August had been having a slight attack of fermental diarrhœa, which began with vomiting, headache, and a slight rise of temperature lasting a few hours. This was soon followed by four or five greenish-yellow discharges in the twenty-four hours, and a normal temperature. The diarrhœa diminished in two or three days, and the child seemed much better, but after a few days she was suddenly attacked with a temperature of  $39.4^{\circ}$  to  $40^{\circ}$  C. ( $103^{\circ}$  to  $104^{\circ}$  F.) and with frequent discharges of mucus and blood. She lost rapidly in weight, and looked very sick. After twenty-four hours, however, the movements became normal; and on the following day, although left weak and prostrated, she seemed perfectly well, and had no return of the attack. During the acute symptoms it seemed as if she were attacked by one of the more severe forms of colitis, but the rapid recovery left the diagnosis very doubtful.

The next case was that of a child (Case 438), seven years old, who entered my wards at the City Hospital with a history of having had a slight diarrhœa for a few days. The temperature was but slightly raised. The movements were infrequent, of a greenish-yellow color, and contained no blood or membrane, and scarcely any mucus. The child seemed fairly well on entering the hospital, but during the following few days became much exhausted. Although no other intestinal symptoms appeared, he sank rapidly, and died apparently from exhaustion.

The autopsy showed extensive lesions of the whole colon, the mucous membrane was greatly thickened, and there were numerous ulcerations.

The third case was that of a boy (Case 439), four years old, who was brought to the Children's Hospital for frequent vomiting following an attack of diphtheria. During the first three weeks that he was in the hospital the vomiting was the chief symptom. He was fed by nutritive enemata and improved in his general strength. Later, however, he became very much emaciated, the vomiting increased in frequency, and a few days before he died there was a slight diarrhœa. The temperature was normal or subnormal during the whole course of the disease.

During the last four or five days the symptoms had pointed almost entirely to the stomach, but the post-mortem examination showed nothing abnormal in the stomach, lungs, heart, kidneys, or spleen. The mesenteric glands were swollen in the region of the ileo-cæcal valve. The walls of the ileum and colon were thickened and reddened. There was a slight deposit of fibrin over part of the mucous membrane of the ileum. The lower 35 cm ( $13\frac{3}{4}$  inches) of the colon were found to be much thickened, the inner surface was of a dark-greenish color, and beneath it the tissue was deeply injected. The thickening seemed largely due to an exudation on the mucous membrane, which could not be torn away. The thickening ended quite sharply, but on some of the valvulæ conniventes above a similar membranous deposit could be found. In the colon the thickening was most marked in the cæcum and the rectum, and least so in the transverse colon, and the process seemed older than in the ileum. Cultures from the various organs were negative. Various organisms were found in the ileum, but none that seemed to be of especial significance.

**ETIOLOGY.**—The etiology of these diseases I have already described under general etiology.

**SYMPTOMS.**—The symptoms of these forms of acute inflammatory ileocolitis vary greatly, as a rule, but in a general way they can be recognized by a group of symptoms which differ from those of the non-inflammatory diarrhœas spoken of as fermental diarrhœa and cholera infantum. The best work which has been done on the symptomatology of these diseases

is that by Holt, but we still find that the symptoms of these different forms of ileo-colitis are very unsatisfactory and unreliable for differential diagnosis.

The onset of the disease may be preceded by a fermental diarrhœa, or it may be acute from the beginning and have prodromal symptoms of no more than a few hours. The temperature is elevated, the pulse is quickened, and the infant loses rapidly in weight and strength. The discharges are perhaps ten or twenty, or even more, in the twenty-four hours. Where the lesions are in the rectum there is tenesmus both before and after the discharge, and in the beginning of the attack an almost continuous desire to have a movement. The discharges contain fæcal matter at first, but soon become small, and consist of mucus, with sometimes pus, blood, and shreds of membrane. The odor may be very offensive, but when the mucus predominates there is very little odor. The color and consistency are extremely variable, but generally the consistency is lessened and the color is a mixture of green, brown, and yellow. The blood is usually from congestion of the blood-vessels and straining, rather than from ulceration. Therefore we cannot determine from the presence of blood whether ulceration is present or not. At first the abdomen may be soft and not tender, but later in the disease it becomes distended, tympanitic, and somewhat tender, especially along the course of the colon. Vomiting may occur at times. In severe cases the child is very restless, and there may be delirium and convulsions. The appetite is usually much lessened. The urine is nearly always lessened in quantity, is high-colored, and sometimes contains a small amount of albumin, especially when the temperature is high. Acute nephritis is, however, rare in these cases. Where there is much tenesmus and straining, and where the discharges are especially frequent, prolapse of the rectum may occur. The discharges often cause great irritation around the anus and on the buttocks.

DIAGNOSIS.—These forms of ileo-colitis are diagnosticated from the fermental diarrhœas by the continued heightened temperature, the more frequent discharges, the small amount in each, the presence of blood or membrane, and the tenesmus. They may be differentiated from cholera infantum by the continuous and excessive vomiting and the serous discharges of the latter disease.

PROGNOSIS.—The prognosis of ileo-colitis, where ulceration has not occurred, is usually favorable, the duration of the disease being a few weeks. Some cases, however, are more severe, and sometimes prove fatal in a few days. Where there is ulceration, the prognosis is rather unfavorable. Where there is a diminution in the frequency of the discharges and fæcal matter begins to reappear, and where the nervous symptoms and exhaustion lessen, the prognosis is good; but where the symptoms increase in severity and the face looks pinched, where intractable vomiting arises and the nervous symptoms predominate, the prognosis is very unfavorable.

The prognosis is less favorable where the ileo-colitis is complicated by



broncho-pneumonia or tuberculosis. It is much influenced by the time of the year at which the attack takes place, the prognosis being worse if the disease occurs at a time when the convalescence is during a long heated period. The prognosis is also worse where the infants have to be treated in crowded cities and in the midst of unsanitary surroundings.

Although there are no symptoms typical of the different forms of acute ileo-colitis, yet their clinical pictures differ somewhat.

It is usually found in the simple catarrhal ileo-colitis, where ulceration has not taken place, that the symptoms are milder and that there is apt to be vomiting. These cases generally begin to improve in one or two weeks, and recover entirely in another week. An intestinal disturbance of a mild character may result, however, and prolong the disease. The children are usually a long time in regaining their strength, and relapses are quite common in this form if the diet is not carefully regulated.

Sometimes, however, simple catarrhal ileo-colitis may be represented by symptoms of a very severe type, and it may run a rapid course, and end fatally.

Where follicular ulceration has taken place the stomach is not apt to be much involved, the temperature is not, as a rule, high, and the course of the disease is rather slow, irregular, and prolonged. The infant fails steadily, and commonly dies. A remission in the symptoms and an improvement in the character of the faecal discharges should lead us to infer that ulceration has not taken place. Where the inflammation is simply follicular, without ulceration, the cases are very apt to recover.

Pseudo-membranous ileo-colitis is rare in infants, but when it occurs it is the most severe of all the forms. I have already stated that it is this form which is usually spoken of as epidemic or sporadic dysentery. The temperature is high,— $39.4^{\circ}$ ,  $40^{\circ}$ , or  $40.5^{\circ}$  C. ( $103^{\circ}$ ,  $104^{\circ}$ , or  $105^{\circ}$  F.). There are apt to be blood and membranous detritus in the discharges. The progress of the disease is usually rapid and without remission, and death may take place in a week or ten days. The nervous symptoms, such as restlessness and delirium, are quite prominent. The diagnosis of this class of cases, as I have just told you, can be made positively only by finding shreds of membrane in the discharges.

**TREATMENT.**—The treatment of these forms of ileo-colitis should usually be in the beginning the same that I have already described for fermental diarrhoea. It may in this sense be spoken of as prophylactic, for in a large number of cases the organisms which produce ileo-colitis find a means of entrance through the irritated mucous membrane produced by a preceding fermental diarrhoea. Where the case is seen in its earlier stages, a mild laxative should be given, in order to clear away, as far as possible, the pathogenic organisms, which are present in large numbers. Small doses of castor oil act most efficiently, and can usually be given, especially to infants, without causing nausea or gastric irritation.

In addition to this treatment by the mouth, thorough irrigation of the

colon should be employed. This should be done twice in the twenty-four hours with warm sterilized water containing 3.75 grammes (1 drachm) of borate of sodium to the pint of water. One or two gallons of water should be allowed to flow in and out of the intestine at each irrigation. After the irrigation, small enemata of thin mucilage, about 120 c.c. (4 ounces), containing 15 c.c. ( $\frac{1}{2}$  ounce) of bismuth in suspension, may be given once in three or four hours.

According to the degree of pain, restlessness, and general discomfort, a slight amount of opium can be given in these injections, but in all cases this drug should be administered with great care; one drop of tincture of opium in the first year, and two drops in the second year, once in five or six hours, will usually be sufficient to make the infant comfortable. The effect of the opium should be carefully watched, and the dose increased or decreased as is necessary.

Where the tenesmus is extreme, it is well to use suppositories containing from 0.015 to 0.03 gramme ( $\frac{1}{4}$  to  $\frac{1}{2}$  grain) of cocaine. These suppositories will often give great relief if the painful lesions are mostly in the rectum, but where the lesions are higher in the colon they are not of much value.

The use of antiseptics by the mouth I do not recommend. Bismuth can be given by the mouth with some advantage in these cases, but the dose must be considerable to accomplish good results. One-half drachm in the twenty-four hours should be given to a child a year old, and for older children the dose should be proportionately increased. Alcoholic stimulants can be given with benefit at all stages of the disease if there is evidence of a weakened heart, or if much exhaustion is present.

A very limited amount of food of any kind should be given during the first twenty-four hours. Sterilized water containing an alcoholic stimulant and barley water had better be given at first, as it has been found that where a sterile liquid is taken by the mouth the number of bacteria in the intestine diminishes rapidly. When a perfectly fresh milk can be obtained it can be used, if sterilized and modified in its various elements so as to be adapted to the digestion of the especial case. A moderate percentage of fat and sugar, such as 3 and 5, and a proteid percentage of about 2, is a very good prescription to begin with. Weak broths can also be given.

In some cases of ileo-colitis, after the acute symptoms have ceased the diarrhoea continues for many months and the disease becomes chronic. In these cases the temperature may be normal, and there is no especial pain or tenderness. The appetite often returns, but the child does not gain in weight, or it loses. The discharges are not so frequent as during the acute stage of the disease, varying from six or eight to two or three in the twenty-four hours. The discharges have a lessened consistency, and contain mucus and undigested food. There may at times be exacerbations of the symptoms, and the children are very apt to die of some intercurrent disease.

The treatment is change of air if possible, and otherwise is essentially



dietetic. The rules which I have already given you in speaking of the treatment of fermental eases are applicable also to this class of cases.

The pathological conditions most commonly found in these chronic forms of ileo-colitis are great thickening of the muscular tissue, pigmentation of the mucous membrane, and very extensive ulceration.

*Amœbic Ileo-Colitis*.—The next form of ileo-colitis which I shall speak of is the *amœbic*. It has its own definite anatomical lesions, which are usually in the colon.

The disease is caused by a well-recognized organism, called the *amœba coli*. It is very rare in northern climates, and is most frequently met with in tropical countries. A frequent source of infection by the *amœba coli* is drinking-water.

I have here a specimen (Plate III., Fig. C, facing page 112) from the intestine of a case of amœbic ileo-colitis. The large round bodies which you see lying in more or less clear spaces are the amœbæ coli. The organism can also be detected by directly examining the discharges under the microscope.

The characteristic pathological lesion of this form of ileo-colitis is the peculiar, undermined condition of the edge of the ulcers and of the mucous membrane. The amœbæ are found not only in the intestine, but also in the various organs, and with especial frequency in the liver.

The disease is usually acute in its onset, but sometimes it may be gradual. The duration may be two or three months.

There are no especial symptoms by which to distinguish this form of ileo-colitis from the others of which I have just spoken, and the only positive proof of the existence of the disease is the presence of the amœbæ in the discharges.

The disease is rare in children, and the prognosis is very unfavorable.

The treatment which has been followed by the most favorable results is, in addition to frequent and thorough irrigation of the intestine, injections of solutions of sulphate of quinine (1 to 5000). This treatment, however, affects only the amœbæ which are in the intestine, and not those which are embedded in the tissues.

*Typhoidal Ileo-Colitis* (Typhoid Fever).—The typhoidal form of acute ileo-colitis is an infectious disease with a definite pathology, and is characterized by constant changes in the lymph-follicles, chiefly at the lower end of the ileum, in the mesenteric lymph-glands, and in the spleen. The disease is produced by the bacillus of Eberth, which is constantly present in the lesions. Infection takes place largely through the gastro-enteric tract. The usual mode of conveyance into the body for the typhoid poison is infected milk or water. Typhoid fever is exceedingly rare in the first two years of life, is uncommon under three years, and after the third year becomes more common as the child grows older.

I have here a specimen (Fig. 125) of the bacillus of typhoid, showing its morphology.

It is about three times as long as it is broad, and is about one-third as long as the diameter of a red blood-corpuscle. It is rounded at the ends.

The pronounced pathological lesions, severe symptoms, and great violence in type which are so characteristic of the typhoid fever of later years are so rare in infancy and early life that I shall confine myself in what I have to say concerning this disease to the conditions which it presents in the latter period.

FIG. 125.



Bacillus of typhoid.

**PATHOLOGY.**—Although the more advanced and severe lesions of typhoid fever may occur in the early as well as in the later years of life, yet its characteristic lesions in young subjects are the milder and less severe pathological changes of the disease. These consist essentially of a hyperplasia of the solitary lymph-follicles and Peyer's patches, and the process, instead of going on to ulceration, usually terminates in early resolution with fatty degeneration of the cells. Hemorrhage and perforation are therefore rare complications in the typhoid fever of early life. There is, however, nothing distinctive of typhoid fever in this hyperplasia of the lymph-follicles in children, for it is not uncommon to find this condition where death has occurred from other diseases of the intestine. It may also be present in such diseases as measles, diphtheria, and scarlet fever. Very marked hyperplasia of the lymph-follicles may be produced in children by irritating substances and by foreign bodies, not only food, but also drugs, such as turpeth mineral. I have, in fact, seen, at the post-mortem examination of a child, marked enlargement of the lymph-follicles caused by doses of turpeth mineral given during life as an emetic. The pathological conditions in typhoid fever in the early years of life may be said to correspond to those which are met with in the aborted forms of the disease in later life.

**SYMPTOMS.**—The stage of incubation of the disease lasts from one to two weeks. The symptoms are, generally, not severe. The prodromal stage is usually short, young subjects having less power of resistance to the



poison than adults, in whom the prodromal stage is often prolonged. As a rule, the temperature is moderate, but it may be high, as in adults, without, however, producing as severe symptoms, since children, commonly, are less affected by a high temperature than adults.

The duration of the disease is generally much shorter than in adults. This short duration depends largely upon the mild form of the intestinal lesions, and usually shows that marked ulceration has not taken place. The temperature chart in this mild typical form of the disease is not apt to be so regular as where the lesions are pronounced. The temperature, although it returns to the normal by lysis, does not show so gradual a lysis as where marked lesions have occurred and where other sources of toxæmia have complicated the disease. The pulse is usually quickened in correspondence to the height of the temperature. The respirations are not especially increased. The nervous symptoms so marked in later life are not prominent in early childhood. Headache slighter than that in adults may occur. Delirium, convulsions, and vomiting may be present. These symptoms, however, are not common. In some cases cerebral symptoms simulating somewhat those of meningitis arise, and are probably due to cerebral congestion or to toxic action. Aphasia occurs rather more frequently in young children than in adults. Its cause is not known, and it appears usually when the disease is declining. It may last for one or two weeks.

The characteristic of typhoid fever in young children, as I have seen it, is apathy. The child takes the nourishment which is given to it, is not especially restless, and usually lies in a half-somnolent condition. As the disease progresses, it gradually returns to a more natural mental condition.

Although it is probable that in most cases of typhoid fever there is some enlargement of the spleen, it is often impossible to detect this change by palpation, and percussion of the spleen in young children is well known to be very misleading. In some cases the rose-colored spots appear on the abdomen, but in quite a number I have been unable to detect them. There is apt to be a slight bronchial catarrh. The bowels are often constipated, though sometimes diarrhœa is present. The tongue is not so likely to be dry as in older subjects, and, although coated, it soon becomes clean at the tip and edges. The abdomen may be somewhat distended and tympanitic, but this symptom is often not marked, and pain and tenderness are rather infrequent. Epistaxis is rare in the typhoid fever of children. There is at times a slight albuminuria during the height of the fever, but a complicating nephritis is rare.

**DIAGNOSIS.**—During the first few days, typhoidal ileo-colitis may often be mistaken for various forms of febrile gastro-enteric disease. A number of acute diseases, such as the exanthemata and pneumonia, may simulate in their prodromal stages those cases of typhoid fever which begin with violent symptoms. The vomiting which occurs in the prodromal stage of typhoid fever may, in connection with the child's apathy, simulate the

early stages of tubercular meningitis. It is therefore often impossible, in the early days of the disease, to make a positive diagnosis, and in some cases we are left in doubt as to the diagnosis for a week or ten days. The characteristic symptoms of the acute diseases already referred to, and of tubercular meningitis, will later be so apparent as to leave the diagnosis no longer doubtful. Jacobi lays stress on the probability of typhoid when there is a continuous high fever which is well borne by the infant, and when the intestinal symptoms are not violent. We should also remember that the differential diagnosis between the typhoidal form of ileo-colitis and malaria is at times, especially in children under two years of age, very difficult, and perhaps impossible, until the blood has been examined. Epidemic influenza may in its onset simulate typhoid fever, but the period of doubt is very short. Acute miliary tuberculosis may in its typhoid type simulate typhoidal ileo-colitis very closely. Where, in the latter disease, the rose-colored spots do not appear, the delirium, distended abdomen, enlarged spleen, and even the irregular temperature, at times common to both diseases, may make the resemblance so close that the differential diagnosis can not be made until the post-mortem examination.

**PROGNOSIS.**—The prognosis of typhoid fever in early childhood is good. The complications, whether arising from local disturbance of the intestine or from cardiac and pulmonary disease, are rare in comparison with those met with in later life. You must remember, however, that the disease varies very much in its severity in different epidemics and in different individuals, and that a child may have a severe type of typhoid fever and die from it.

**TREATMENT.**—The treatment of the typical mild form of typhoid fever in young children is to keep the child in bed and to feed it regularly every two or three hours with fresh milk, modified according to the condition of its digestion, and prepared by heating to  $75^{\circ}\text{C}$ . ( $167^{\circ}\text{F}$ .). As a rule, antipyretic drugs should not be used. The child should be bathed at least twice a day with water at a temperature of  $32.2^{\circ}\text{C}$ . ( $90^{\circ}\text{F}$ .), not necessarily for the purpose of reducing the temperature, but as a hygienic measure. In most of the cases which I have seen this is all the treatment that has been found necessary from the beginning to the end of the disease. Where there are great restlessness and delirium, with a high temperature,  $40.5^{\circ}$  to  $41^{\circ}\text{C}$ . ( $105^{\circ}$  to  $106^{\circ}\text{F}$ .), baths should be given once every three or four hours, but the temperature of the water should not be below  $26.6^{\circ}$  to  $29.4^{\circ}\text{C}$ . ( $80^{\circ}$  to  $85^{\circ}\text{F}$ .), as this is usually sufficient to allay the symptoms. If the temperature remains high and there are symptoms of serious import, such as occur in the advanced stages of the adult type of typhoid fever, colder water can be used; but, as a rule, it is not wise to employ water of a low temperature in children to the extent to which it has been found useful in adults. Alcoholic stimulants should be given where there are signs of exhaustion.

The various complications which arise should be treated symptomatically.



The same care should be exercised during the convalescence of the child as in the advanced convalescence of the adult.

I have here in the wards a case of typhoid fever.

This child (Case 440), a boy, is five years old.

Five days before entering the hospital he was taken sick with general malaise and fever. There had been no other symptoms, such as epistaxis or vomiting. On entering the hospital, an examination showed the tongue to have a thick brownish coat in the centre and a thin coat on the tip and edges. The child was in an apathetic condition. The pulse was rapid and regular. Nothing abnormal was found in the thorax. The abdomen was distended and tympanitic, and showed one rose-colored spot. The spleen could be easily felt

#### CASE 440.



Typhoidal ileo-colitis. Male, 5 years old.

2.5 cm. (1 inch) below the border of the ribs, and on percussion the dulness reached as high as the seventh rib in the axillary line. I have marked this enlargement of the spleen and the lower border of the ribs in black. The upper border of the splenic dulness is marked by a broken line, and the figure 7 marks the seventh rib. The blood showed no leucocytosis. You see that the pupils react equally to light. You will also notice the apathetic expression of the child's face, and that he takes very little notice of anything. An examination of the urine shows the color to be normal, the reaction neutral, the specific gravity 1026, and that there is a slight trace of albumin. The sediment shows occasional hyaline and fine granular and fibrinous casts.

(Subsequent history.) On the third day after entering the hospital, the eighth day of the disease, the child became very stupid, and sometimes delirious. There was a slight cough.

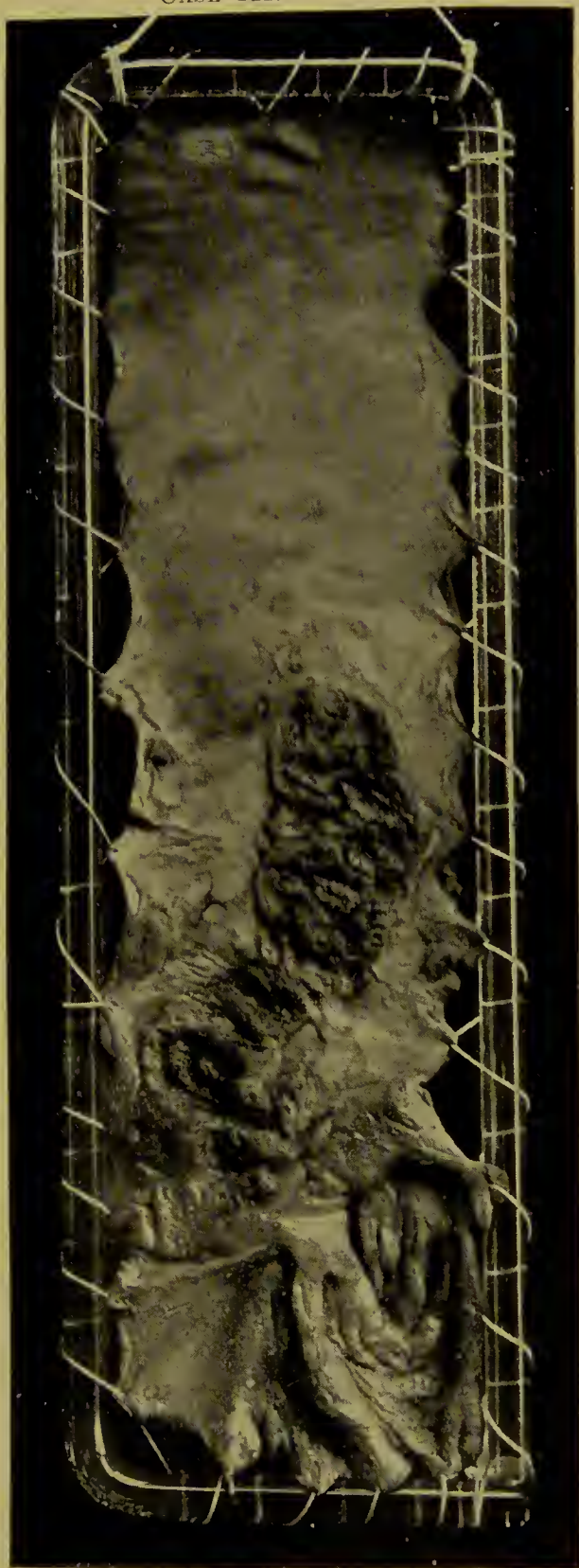
On the twelfth day of the disease the child cried out at times, and was delirious. The skin was dry and hot. There were no more rose-colored spots. There seemed to be slight tenderness in the lower iliac fossa, but there was no gurgling.

On the fifteenth day of the disease the temperature began to fall by lysis, and the child began to be fretful.

On the eighteenth day the temperature became normal.

By the twenty-first day the child seemed bright, and was playing with its toys. The pulse was stronger. One week later it was sitting up in bed, and had a strong pulse and a good appetite. A few days afterwards it was up and about the ward, perfectly well. Here is the chart (Chart 31, page 995) from the fifth day of the child's illness until convalescence was established on the twenty-fifth day.

CASE 441. FIG. 126.



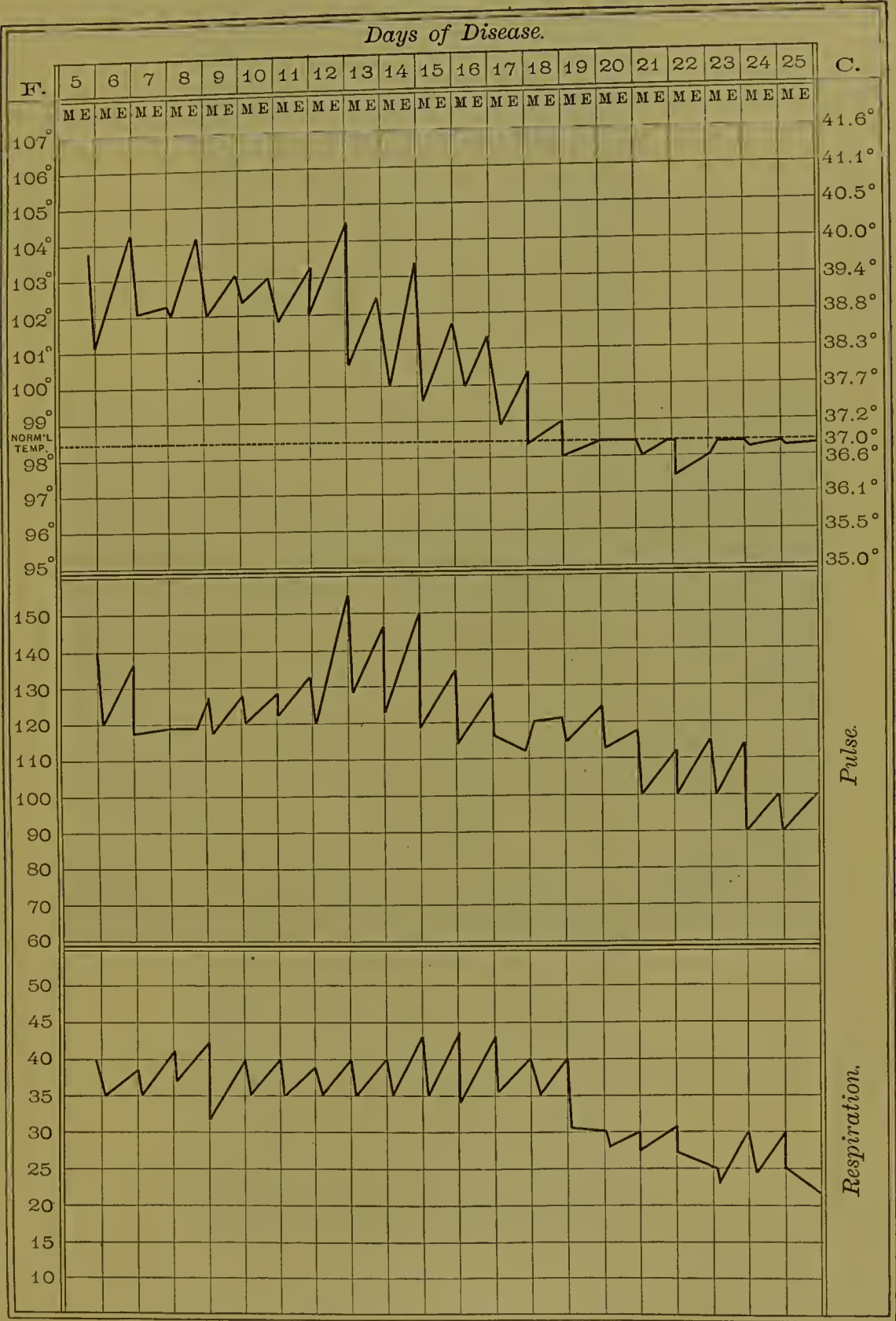
Typhoidal ileo-colitis, showing ulcers of colon. Female, 2 years old.  
U. S. Army Medical Museum.





Through the kindness of Dr. Billings I am enabled to show you this intestine (Fig. 126), which was taken from an infant with typhoidal ileo-colitis.

CHART 31.



Typhoidal ileo-colitis. Male, 5 years old.

The infant (Case 441) was a patient of Dr. S. S. Adams, of Washington. In this case the irregularity of the temperature curve and the prominent symptoms of cerebro-spinal



irritation rendered the diagnosis so obscure that typhoid fever was not suspected until a few days before death. The post-mortem examination showed marked congestion of the entire brain, chiefly on the right side. The left hemisphere was covered with a gluey substance which filled the sulci and was especially abundant around the Sylvian fissure. The heart was normal. The lungs showed marked hypostatic congestion. The liver was normal. The gall-bladder was empty and pale. The spleen was enlarged. The kidneys were normal. The stomach was congested. The mesenteric glands were enlarged and soft. The intestines contained a quantity of yellowish watery feces. The lesions which you see in this specimen are in the ileo-colic portion of the intestine. You see that there is thickening and ulceration of Peyer's patches, and to a less extent of the solitary follicles.

In order to impress upon you that in infants swelling of Peyer's patches and of the solitary follicles is not distinctive of typhoid fever, and that this condition frequently occurs from irritations of various kinds, I show you under this microscope (Fig. 127) a section taken from the intestine of a child.

FIG. 127.



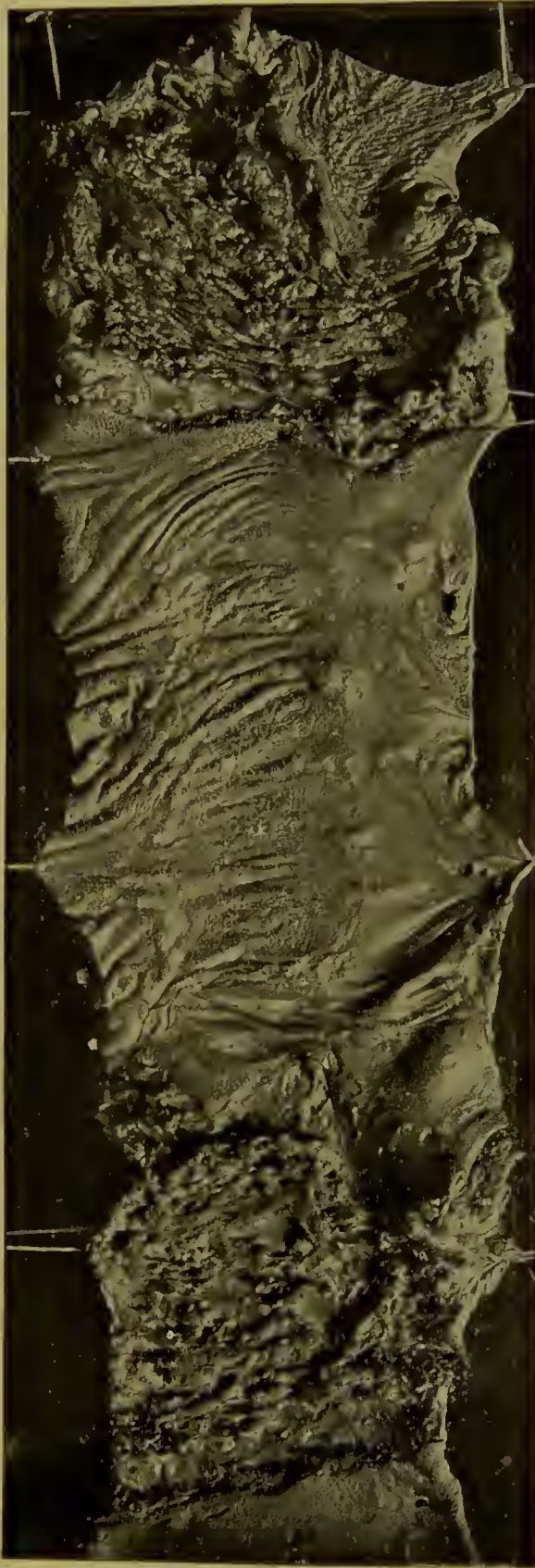
Enlarged Peyer's patches closely simulating the lesions of typhoidal ileo-colitis. Muc. Mem., mucous membrane; Fol., enlarged follicles; Mus., muscle.

The macroscopic appearances of this intestine so closely simulated the early stage of typhoid fever that cultures were made from it by Dr. Prudden to settle this question. No bacilli were found. This condition is often found in children in acute non-typhoidal ileo-colitis.

**CHRONIC ILEO-COLITIS.**—Under chronic ileo-colitis we include chronic appendicitis, those forms of ileo-colitis which follow acute attacks of ileo-colitis, and tubercular disease of the intestine. I have already spoken of chronic appendicitis and the chronic form following acute ileo-colitis, and shall, therefore, devote only a few words to intestinal tuberculosis.

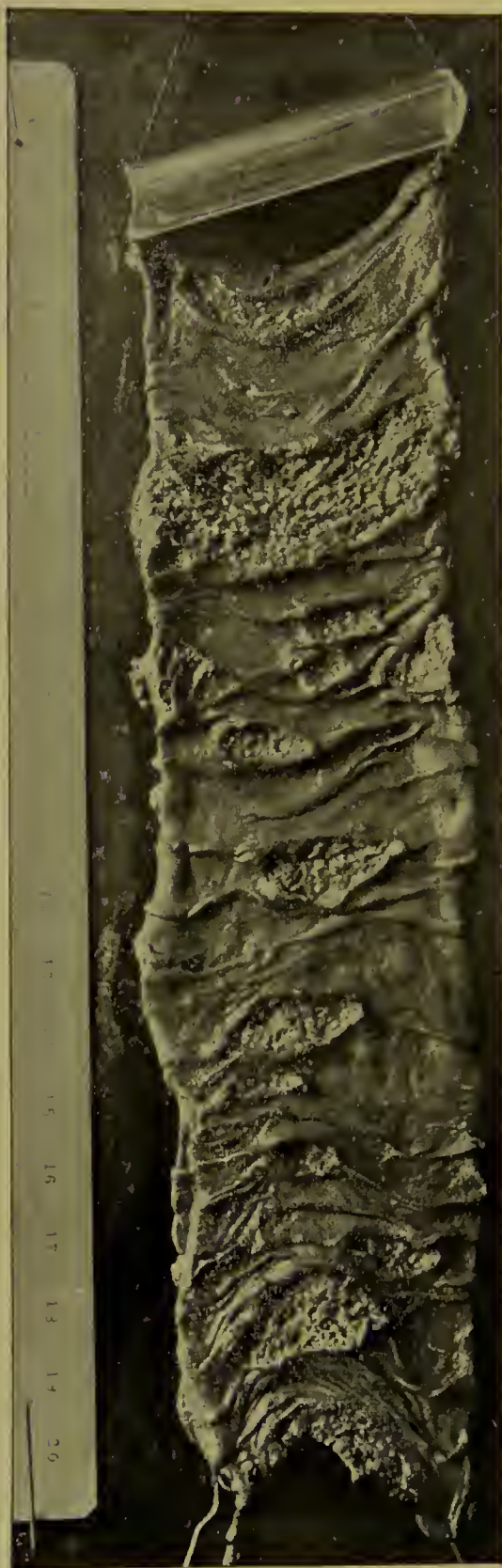
**Tubercular Ileo-Colitis.**—Tubercle of the intestine in infancy and childhood is quite common. The small intestine is most frequently involved. The disease may be primary in the intestine, but this is very

CASE 442. FIG. 128.



Tubercular ulcers of colon. Female, 8½ years old. Museum of the College of Physicians and Surgeons, New York.





Tubercular ulcer of small intestine. Female,  $2\frac{3}{4}$  years old. Museum of the College of Physicians and Surgeons, New York.

rare. At the Boston Children's Hospital I have had one case where the tubercular lesions were confined to the intestine and to the mesenteric glands. In this case Professor Councilman considered that the evidence was in favor of the intestinal tubercle antedating the tubercle of the glands. In the great majority of cases the tubercular ileo-colitis is secondary to tuberculosis elsewhere, and in such cases frequently follows tuberculosis of the mesenteric glands.

**PATHOLOGY.**—According to Osler, the ulcers are situated chiefly in the ileum, and involve the solitary follicles and Peyer's patches. The tubercular ulcer has the following characteristics. In contradistinction to the typhoidal ulcer, the long diameter of which coincides with the long axis of the intestine, the tubercular ulcer is transverse to the long axis, rarely ovoid, and often irregular in outline. The edges overhang, and the base is infiltrated.

Through the kindness of Professor Northrup, I have here some specimens of tubercular ulcers. This first specimen (Fig. 128, facing page 906) was taken from a female (Case 442) eight and one-half years old.

This child, a patient of Dr. Northrup's, was attacked, two months before her death, with chills, fever, and prostration. The temperature at first varied from 38.8° to 39.4° C. (102° to 103° F.), but as the disease progressed the temperature gradually fell. There was rapid emaciation. The abdomen was sunken at first, but later became tense. There were pain, tenderness, and resistance in the right inguinal region. The submaxillary, cervical, and inguinal lymph-glands were enlarged. The urine contained albumin and hyaline casts. There was diarrhoea. The autopsy showed the lungs to be normal. The bronchial and retroperitoneal lymph-glands were enlarged and cheesy. The colon shows two large, sloughing ulcers, one in the region of the cæcum and the other in the ascending portion. You see that they are transverse to the axis of the colon, and that their edges are overhanging. The entire membrane is thickened, and there is some follicular ulceration.

This next specimen (Fig. 129) was also taken from a patient of Dr. Northrup's.

The child (Case 443), a female, two years and eight months old, had had diarrhoea occasionally for a year. It had also had convulsions. It died soon after entering the hospital. The autopsy showed these extensive ulcerations in the small intestine (I.) and this large ulcer in the cæcum (II., page 908). There were also tubercular ulcers in the middle third of the colon.

The peritoneal surface showed miliary tubercles. The mesenteric and bronchial lymph-glands were markedly enlarged and cheesy.

**SYMPTOMS.**—The symptoms of tubercular ileo-colitis are varied and rather indefinite. The most common symptom is a persistent diarrhoea. The diarrhoea does not, however, correspond to the extent of the lesions, as large ulcers may exist and constipation be present, especially if they are in the ileum. In cases of primary tubercle of the intestine the only sure means of determining the tubercular character of the disease is the finding of the bacillus tuberculosis in the discharges. Where the disease is secondary to tuberculosis elsewhere, the tubercular involvement of the intestine may



be suspected when at any time during the course of the disease the infant is attacked with diarrhœa of an obstinate nature. In these cases the diagnosis can also be established by finding the tubercle-bacilli in the discharges.

CASE 443. FIG. 129.

II.



Large tubercular ulcer of cæcum.

The prognosis is very unfavorable, and death may occur either from the severity of the intestinal symptoms or, as pointed out by Osler, more rarely by perforation or hemorrhage.

**ANIMAL PARASITES.**—The animal parasites which are found in the intestines of infants and children are the same that occur in older subjects. The only ones, however, which are common and important enough to speak of are the *oxyuris vermicularis* (pin-worm), the *ascaris lumbricoides* (roundworm), the *tænia solium*, and the *tænia mediocanellata*.

**OXYURIS VERMICULARIS.**—The *oxyuris vermicularis* is a minute worm which looks like a little piece of white thread. The female is from 0.6 to 1.2 cm. ( $\frac{1}{4}$  to  $\frac{1}{2}$  inch) in length. The male is about one-third as large, and has the tail rolled into a spiral.

I have here some of these worms to show you (Fig. 130). Their de-

velopment takes place in the large intestine, and the mature worms deposit their eggs in the rectum. They enter the intestine through the mouth, and children are very apt to reinfect themselves by carrying the eggs on the fingers or under the nails to their mouths.

FIG. 130.

*Oxyuris vermicularis.* *Ascaris lumbricoides.*

These worms sometimes exist in large numbers, and their development is so rapid that it is often difficult to dislodge them completely. The most common symptom of the oxyuris is an intense itching about the anus. The sleep of the child is disturbed by this irritation, and various nervous symptoms develop in children who are infested with this parasite. Thus incontinence of urine sometimes results. In girls the parasite, by migrating from the anus to the vulva, may cause a vulvo-vaginitis.

DIAGNOSIS.—The diagnosis of the presence of these, as of other intestinal parasites, can be made only by finding the worm or its ova. Where they are suspected, an enema of clear water should be given. If the parasites are present, they will be dislodged, and careful inspection will disclose their presence. Wherever there are symptoms of reflex irritation in the neighborhood of the anus or the genital organs, the oxyuris should be suspected and sought for. The parasites can often be found in the faecal dis-



charges, and in some cases they can be seen by simply stretching open the anus and examining the mucous membrane of the rectum.

**TREATMENT.**—Although most of the worms are in the rectum, yet they also infest the upper parts of the intestine, and therefore cannot be reached by enemata. In many cases enemata of salt-and-water are sufficient to produce a cure, but in some cases the salt, even in small amount, is so irritating that it cannot be used. Infusions of quassia may also be employed as enemata. One of the most effective methods of dislodging the parasite is to give every evening at bedtime an injection of 60 c.c. (2 ounces) of sweet oil. This is allowed to remain in the rectum for five or six minutes, and a large enema of water is then used to wash out the oil, which usually carries with it the parasites from the lower colon and the rectum. Care must be taken in regard to cleanliness, so as to prevent reinfection.

Where this treatment is not sufficient, lozenges of santonin, 0.01 to 0.03 gramme ( $\frac{1}{8}$  to  $\frac{1}{2}$  grain), according to the age, may be given two or three times daily.

Every two or three days a cathartic, such as castor oil or calomel, should be given. Care must be used in giving santonin not to produce symptoms of poisoning, such as gastro-enteric irritation, dizziness, and yellow vision. This occurrence, however, will not be common if in each case you carefully watch the effect of the drug on the child. You must also bear in mind that serious symptoms, such as convulsions, have been caused by a lack of care in using this drug in young children.

Under this treatment, aided by high rectal injections, the worms can in most instances be eradicated. I have, however, met with very intractable cases where months and even years had elapsed before treatment of any kind was successful. In such cases temporary relief can be obtained by giving the child each night, or two or three times a week, a small enema of oil.

**ASCARIS LUMBRICOIDES.**—The ascaris lumbricoides is a long, cylindrical, yellowish-white or reddish-yellow worm, pointed at both extremities. The male is distinguished from the female by the fact that it is smaller and is always rolled upon itself, while the female is straight. The length of the male is from 10.4 to 18 cm. (4 to 7 inches), and that of the female from 15.5 to 28.5 cm. (6 to 11 inches).

Here are some specimens (Fig. 130, page 909) of lumbricoid worms. The larger worm is the female. The eggs of this worm are oval in shape, 0.075 mm. long and 0.058 mm. wide. When they are first passed they are almost transparent, but they soon become yellowish and opaque. These eggs are not developed within the intestine, but may pass out with the feces. They are very tenacious of life, and may develop under favorable circumstances after many years. The embryos are developed outside of the body, and reach the intestine with the drinking-water, where they develop into the mature worm.

The habitat of the worm is usually in the small intestine. It may,

however, pass through the rectum either with the *faeces* or alone, and may migrate into the stomach, *oesophagus*, or nose. Instantaneous death has resulted from the entrance of these worms into the air-passages. They may also at times enter the common and cystic bile-duets, and they have even penetrated farther and caused abscess of the liver. There is no danger of their perforating a normal intestine, but where ulceration has been present perforation has occurred.

**SYMPTOMS.**—There are no especial symptoms produced by this worm, and we can diagnosticate its presence only by seeing it or by finding the eggs in the *faecal* discharges. The worm may in some instances produce a feeling of discomfort or even colic in the region of the umbilicus. Neither of these symptoms, however, can be depended upon, and an anthelmintic is required to determine whether the parasite is present. As a rule, the presence of these parasites in the intestine, unless in very large numbers, is not especially important.

**TREATMENT.**—The most efficacious treatment of this form of parasite is with *santonin*, which should be given in the same doses and with the same caution as I have already described in speaking of the treatment of the *oxyuris*.

In addition to *santonin*, the freshly prepared fluid extract of *spigelia* and *senna*, in doses of half a teaspoonful for a child two years old, and a teaspoonful for older children, can be given two or three times a day, care being taken not to produce too much irritation. The oil of *chenopodium*, three or four drops on sugar for a child two or three years old, and eight or ten drops for older children, can also be given. A cathartic should be used in connection with these drugs, as well as with *santonin*.

**TÆNIÆ (Tape-worms).**—Two forms of *tæniæ* occur in children. One of these is the *tænia solium*, the pork tape-worm. It has a slight projection at the apex of its head, around which are a series of hooks, and below which are four sucking-disks. The other form is the *tænia mediocanellata*, the beef tape-worm. It has a blunter head than the *tænia solium*, and does not have the circle of hooks.

There is nothing especial to be said concerning these worms, and I refer to them merely because at times they occur in early life. They are never met with in nursing children when milk forms the exclusive diet. There are no especial symptoms produced by this worm, and the diagnosis is made entirely by finding the segments in the *faeces*. There is no especial danger to life from the presence of the tape-worm.

I have here two specimens (Fig. 131, I., II., page 912) of *tænia* to show you.

The worm in the bottle was from a child from whom the entire worm was expelled, and you can see, by means of a magnifying-glass, the head. The absence of hooks shows it to be the variety called *tænia mediocanellata*. I show you the other worm in order to impress upon you the importance of obtaining the head. You see that the head is not present, and



that it has evidently broken off near the extremity of the neck. In this case the head remained in the intestine and the worm grew to the usual length again. These worms vary in length from 605 to 1512.5 cm. (20 to 50 feet).

The treatment employed for expelling this worm is the same in children as in adults, but we should be very careful not to irritate too much the sensitive gastro-enteric mucous membrane of the young child. The child should first be treated with laxatives, so as to free the intestine. Food

FIG. 131.



Tæniæ. I., without head ; II., with head.

should be withheld from the early evening until as late as possible the next day. An anthelmintic should then be given, followed in one or two hours by a cathartic. This usually results in the expulsion of a large mass of segments. Great care should be taken to prevent the head from breaking off before it is expelled. The anus should be carefully dilated during the expulsion of the worm. Sitting on a vessel of hot water seems to help to prevent the head from breaking off.

There is no anthelmintic which I have found especially successful in expelling the *tæniæ*. One of the most harmless is the alkaloid pelletierine from pomegranate. One-half teaspoonful of the tannate of pelletierine can be given to a child from three to five years old. As dizziness and headache are sometimes complained of, it is well to have the child kept in bed and lying down until the effect of the anthelmintic has passed off. The oleo-resin of male fern may also be used. The dose is 0.94 to 1.88 gramme ( $\frac{1}{4}$  to  $\frac{1}{2}$  drachm). The cathartic which is most useful in these cases is Epsom salt, 7.5 to 15 grammes (2 to 4 drachms).

It is hardly worth while to mention the other numerous anthelmintics which have been recommended, as they are usually inefficient.



## DIVISION XIV.

### DISEASES OF THE LIVER, PANCREAS, SPLEEN, AND PERITONEUM.

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#### LECTURE XLVI.

**LIVER.**—In infants and young children the liver is proportionately larger than in later life. In a previous lecture (Lecture IV., pages 121, 122, 124) I have shown by percussion the size of the liver at different ages, and I shall therefore now merely refer you to what I said at that time.

**ICTERUS.**—Icterus is a symptom of a number of diseases, as well as of disease of the liver, but it so commonly occurs where the liver is either directly or indirectly affected that it is best spoken of in connection with hepatic disease. The icterus which arises at birth, either of the temporary form, such as icterus neonatorum, or from obliteration of the bile-ducts, I have spoken of in a previous lecture (page 107). I have also spoken of icterus as a symptom when describing acute and chronic duodenal indigestion. You must not assume that there is necessarily hepatic disease because icterus is present, as any slight mechanical disturbance in the liver produced by diseased conditions elsewhere may cause icterus. In these cases, even though the liver may be somewhat enlarged, it is not a symptom of much import, and the liver is soon restored to its normal condition, provided that the original disease has disappeared or has ceased to produce hepatic disturbance. Icterus may also occur as a symptom in septic inflammation of the umbilical vein. In these cases the liver is apt to be enlarged and tender. Convulsions commonly occur. Vomiting, diarrhœa, abdominal swelling, pain, and tenderness are present. The temperature is high. The respirations are increased, and death usually occurs from exhaustion or from septic inflammation of the pleura, pericardium, or other parts.

**DISEASES OF THE LIVER.**—Diseases of the liver are not common in infancy and childhood, as the inciting causes of hepatic disease are usually not present in early life. When hepatic disease occurs, it is commonly secondary to some general disease, and therefore it need not be dwelt upon at length in a separate lecture.

The acquired pathological lesions which occur in the liver in infancy

and childhood do not differ from those which are met with in later life. A rapid increase and decrease in the size of the liver are not infrequently met with in disease, and careful measurements have shown that even a very slight disturbance of health may cause in young children a variation of from 2 to 4 cm. ( $\frac{3}{4}$  to  $1\frac{1}{2}$  inches) in the size of the liver.

**ACUTE YELLOW ATROPHY OF THE LIVER.**—It is uncommon for the liver to be decreased in size, but this occurs in the rare cases of acute yellow atrophy at times met with in children. The disease is insidious in its onset, and is characterized by general symptoms of malaise, with icterus and bile-stained urine. In the beginning of the disease the liver is enlarged, but in the later stages it is decidedly diminished. Cerebral symptoms and vomiting are quite prominent, and death invariably occurs.

In most diseases which are accompanied by hepatic disturbance it is much more common to find the liver enlarged than to find it diminished in size. This enlargement may occur from a number of causes, among which is *mechanical congestion*, arising in the course of cardiac disease. I shall presently show you, when speaking of diseases of the heart, a case (Case 503, page 1042) illustrating this form of enlargement.

The morbid conditions of the liver which are most commonly met with are *fatty infiltration*, *amyloid infiltration*, *tuberculosis*, and *interstitial hepatitis*. This latter form of hepatic disease may arise under various pathological conditions. Thus, it is a frequent lesion in syphilis, and may occur in a number of systemic diseases. It may also result from the use of alcohol, and at times it occurs apparently unassociated with disease of any other organ.

Other pathological conditions of the liver, such as hepatic abscess, parasites, such as hydatids, and new growths, such as carcinoma, adenoma, and, as occurred in a case at the Boston Infant Hospital, sarcoma, are too rare to be considered in a general clinical lecture on children.

**FATTY INFILTRATION OF THE LIVER.**—Fatty liver in early life does not differ pathologically from that which is met with at a later period. The liver may or may not be enlarged, and there are no especial hepatic symptoms which characterize this condition, the symptoms being those of the general disease from which the child is suffering. It may be found associated with a number of diseases, especially rhachitis and tuberculosis. When the liver is enlarged from this cause its surface is found to be smooth and palpation is painless.

The prognosis, unless the disease is dependent upon some incurable disease elsewhere, is fairly good.

The treatment is essentially dietetic and hygienic.

**TUBERCULOSIS OF THE LIVER.**—Tuberculosis of the liver occurs in connection with general tubercular disease of other organs, and does not in itself present any especially characteristic clinical manifestations. The disease is commonly found in the form of miliary tubercles and cheesy nodules. Except in rare instances where large caseous masses cause obstruction and



later disintegration of the tissues, with hepatic enlargement and abscess, it is not usually recognized during life.

**AMYLOID LIVER.**—When amyloid changes are present in the liver, other organs, such as the spleen, kidneys, and intestine, are involved. Amyloid infiltration may occur in the course of tuberculosis, where there is chronic disease of the bones, with extensive suppuration, and in wasting diseases. A very prominent symptom in this condition is extreme anæmia. The liver is, as a rule, very much enlarged, and commonly more so than in any of the other hepatic disturbances. Its surface is smooth, and there is rarely hepatic tenderness or pain. Ascites is rare, and there is usually no icterus.

The diagnosis is not difficult if we find that the child has one of the diseases which I have just mentioned as being the causes of amyloid changes.

When these changes occur in the liver the prognosis is very grave, and there is no treatment which will be of more than temporary benefit. The treatment, therefore, is simply symptomatic.

This boy (Case 444) is seven and three-quarter years old.

CASE 444.



Amyloid liver. Pulmonary tuberculosis. Male,  $7\frac{3}{4}$  years old.

There is no history of tuberculosis in his family. He had pertussis when he was one and a quarter years old, and measles when he was three years old. He seemed well and strong until seven months ago, when he became listless and began to have fever and to perspire profusely. Four weeks ago he began to vomit occasionally, to complain of headache, and to cough. You see that although he has evidently lost in weight he is not especially emaciated. His entire skin is extremely pale and has a waxy look, which is apparently not due to jaundice. His mucous membranes show much anæmia. His tongue is heavily coated, and his breath is offensive. He is dull and apathetic. The cervical glands are enlarged and slightly tender, but do not fluctuate. The glands are moderately enlarged in the axillæ and groins. The percussion of the right lung, especially at the apex, is dull, and there are numerous râles. The area of cardiac dulness is not enlarged, but there is a slight systolic murmur at the apex. The spleen is slightly enlarged. The edge of the liver can

be felt below the line of the umbilicus. The area of hepatic dulness is increased, as is represented by this broken line. I have also indicated the lower part of the sternum and the lower borders of the ribs by black lines. There is no hepatic tenderness, and the child does not complain of pain. The lower part of the abdomen is dull on percussion as high as the line which I have drawn under the umbilicus. This is due to a slight amount of ascites. The legs are swollen. The urine has a specific gravity of 1010, and contains a slight trace of albumin, an occasional hyaline cast, and renal epithelium. The temperature has varied from 37.2° to 39.4° and 40.5° C. (99° to 103° and 105° F.). The increased size of the liver is probably due to amyloid infiltration.

(Subsequent history.) A few days later the child grew rapidly weaker, and died of exhaustion.

**INTERSTITIAL HEPATITIS (Cirrhosis).**—The syphilitic form of hepatitis as it occurs in infancy I have already described in my lecture on Hereditary Syphilis (page 489).

Interstitial hepatitis as it occurs in childhood may be *atrophic* or *hypertrophic*. The general symptomatology differs but little from that of the adult. In the beginning the symptoms are very apt to be confounded with those of simple congestion arising from digestive disturbances. There may be abdominal pain, slightly augmented by pressure. Diarrhoea and constipation alternate. There are usually ascites and slight jaundice, and at times dilatation of the subcutaneous abdominal veins. Stigmata composed of collections of dilated minute veins are sometimes observed on the face. The temperature is irregular. As a rule, it is not much heightened, and in fact is often subnormal.

The prognosis and treatment in early childhood are the same as in adults. A certain number of cases seem to have followed scarlet fever and measles.

Alcohol is sometimes an etiological factor in infancy and early childhood. Where the disease is caused by alcohol the pathological condition is, as a rule, atrophy. Enlargement is not common, and the symptoms are the same as in the adult, the ascites being especially prominent.

Where the hepatitis is apparently not dependent on disease elsewhere, and is not due to alcohol, there are no characteristic symptoms beyond the enlargement of the liver. In this form the ascites is usually small in amount, and the diagnosis can be made only by eliminating the other forms of enlargement.

I have here a case of hepatic enlargement which seems to represent clinically that form of hepatic disease which is commonly spoken of as hypertrophic cirrhosis.

This little girl (Case 445, page 918) is eighteen months old.

There is no history of syphilis or of tuberculosis. She had pertussis when she was ten months old, and the cough lasted for several months. She has never taken alcohol in any form. She was well until three months ago, when she began to complain of pain in the abdomen and to become pale. Two weeks before entering the hospital she had diarrhoea, and her abdomen was noticed to be swollen. On entering the hospital and being placed on a proper diet, the diarrhoea ceased, but the swelling of the abdomen increased. The child, as you see, is well developed, but pale. The abdomen is much enlarged. The edge of the liver can be felt nearly as low as the line of the umbilicus. I have marked out the



area of dulness on percussion with black lines. The lower one shows the notch between the right and the left lobe, which is distinct and easily palpable. There is no especial tenderness on pressure. The spleen is slightly enlarged. In the lower part of the abdomen there is a moderate amount of dulness and fluctuation, showing the presence of fluid. There are no glandular swellings. The heart is normal, but is pushed up somewhat by the abdominal distention. I have indicated the cardiac area of dulness by a black line, and the lower border of the ribs and ensiform cartilage by a broken line.

CASE 445.



Hypertrophic cirrhosis. Female, 18 months old.

The child has improved in its general health since entering the hospital, and has a fair appetite. Physical examination shows the presence of no other disease. Without an autopsy, however, the diagnosis must necessarily be held in abeyance.

(Subsequent history.) The child remained in the hospital for a few weeks, and improved in its general health so that it seemed quite bright. The ascites did not increase in amount, but the liver remained enlarged. The child was taken away from the hospital, and its subsequent history could not be obtained.

**PANCREAS.**--Diseases of the pancreas are practically unknown in infancy and childhood, with the exception of the general tissue-changes which may be met with in syphilis, and which I have already described (page 490). New growths of a malignant nature have been reported.

**SPLEEN.**--The spleen may be involved in tuberculosis, and may show amyloid changes in connection with other organs.

It is frequently enlarged in the course of a number of diseases which I have described elsewhere.

**PERITONEUM.**--Diseases of the peritoneum may be of non-inflammatory or inflammatory origin.

The *non-inflammatory* diseases are mostly represented by new growths. These may be of a malignant nature, such as carcinoma and sarcoma, or they may be lipomata or of a cystic character. In this connection it is well to say that tumors of the omentum are rare, but that cysts and hydatids may occur in this region.

The differential diagnosis of these various forms of peritoneal and omental growths can scarcely be made during life.

The treatment is essentially surgical.

The *inflammatory* diseases of the peritoneum are represented by peritonitis.

PERITONITIS.—Inflammation of the peritoneum may be acute or chronic, and is a condition of great importance in infancy and early life. Peritonitis may occur in the infant and child as it does in the adult. It is so rare in infancy and childhood as an idiopathic disease that the cases in which it has been studied post mortem have occurred almost exclusively during uterine life. Many of these, moreover, have presented a history of syphilitic infection. The septic form of peritonitis is not infrequently met with in the early weeks of life, and I have already referred to it when speaking of phlebitis umbilicalis (page 425).

I have here an infant (Case 446) who was brought to the hospital yesterday to be relieved of extreme distention of the abdomen.

CASE 446.



Probable intra-uterine peritonitis. Infant, 5 weeks old.

The infant weighed at birth 4500 grammes (10 pounds), seemed strong, and nursed for three weeks. There is no history of syphilis or of tuberculosis. After birth it began to be icteric. It passed meconium, but the faecal movements since then have always been white. A few days after birth the abdomen began to swell, and it has since continued to increase in size. The skin is very tense, and the veins connected with the portal circulation stand out in marked relief all over the abdomen. The infant vomited once four days ago, and again this morning. It has become much emaciated. There is distinct fluctuation in every part of the abdomen, and dulness on percussion. An examination shows the heart and lungs to be normal.

(Subsequent history.) Laparotomy was performed by Dr. Lovett on the following day. On opening the abdomen a stream of pale fluid was thrown into the air with consider-



able force. A quart of this fluid was removed, and was examined by Dr. Whitney. It proved to be a jaundiced aseptic fluid, but the examination did not reveal its cause. It had a specific gravity of 1016, and contained  $2\frac{1}{2}$  per cent. of albumin. It also showed bile pigment. The sediment contained much blood, many red corpuscles, an occasional white corpuscle, and fat-corpuscles. The flocculi were composed of finely granular material showing in places cells in a state of fatty degeneration.

After the fluid was evacuated a digital examination showed extensive adhesions in the intestine, especially on the under surface of the liver, where nothing but a matted mass could be found. On the supposition that there was some obstruction to the flow of bile into the intestine, an attempt was made with the finger to free the intestine from the lower surface of the liver.

The child rallied well from the operation, and thirty-six hours later a small spot of yellow bile appeared in one of the white movements. After this time bile was passed regularly and the movements became normal. The child's general condition and its nutrition improved.

At the end of ten days it was taken home, but it soon began to fail, and after three weeks died of malnutrition. No autopsy was allowed.

**Acute Peritonitis.**—Infants and children of any age may be attacked by acute peritonitis. It may occur in cases of tuberculosis, of the infectious diseases, of syphilis, and, most frequently of all, of appendicitis. The disease in any of the above forms is exceedingly rare between the ages of six weeks and two years. Where some definite cause, such as one of those just enumerated, cannot be found, the diagnosis is at times difficult from a want of prominence of some of the symptoms, such as the tympanites.

**PATHOLOGY.**—The pathological manifestations in acute peritonitis are reddening and loss of the normal glistening appearance of the peritoneum, soon followed by an exudation varying from a serous to a thick fibrinopurulent character. This exudation glues the coils of intestine together, forming adhesions, which, however, can be readily separated without the aid of a knife.

**SYMPTOMS.**—The symptoms of acute peritonitis vary according as the process is general or localized. The localized form of peritonitis corresponds in its symptoms to what I have already described in speaking of appendicitis, which is its most frequent cause. In general peritonitis the symptoms in infants, as I have already stated, are often obscure. In children the symptoms are usually pronounced and characteristic. The child is attacked with abdominal pain and with general abdominal tenderness. The abdomen becomes distended and tympanitic, and the child assumes the position which will most relax the abdominal walls,—that is, with the thighs flexed and the knees bent. Vomiting is very apt to be present, and is augmented when food is given. The bowels are often constipated, although at times there may be diarrhoea. The temperature is usually high,  $38.3^{\circ}$  to  $40.5^{\circ}$  C. ( $101^{\circ}$  to  $105^{\circ}$  F.); in some cases, however, the temperature may be normal or subnormal. The pulse is small and rapid. The respirations are not only accelerated, but also superficial, as deep respiration causes pain. The face has an anxious expression, and shows great suffering. Where recovery takes place, these symptoms gradually subside after a few days, the tenderness, pain, and tympanites disappear, and the child's face assumes a tranquil

look. When improvement does not take place, the pulse becomes weaker and quicker, the breathing more superficial and rapid, there is chilling of the extremities, and the child dies usually within a week.

**PROGNOSIS.**—The prognosis in these forms of acute general peritonitis is always serious. Constant vomiting makes it especially grave.

**TREATMENT.**—In treating cases of acute general peritonitis when seen in the early stages, a saline, such as sulphate of magnesium, can be given in doses of 1.87 c.c. to 3.75 c.c. ( $\frac{1}{2}$  to 1 drachm), according to the age of the child. When, however, the disease is more advanced and there is great pain, opium will have to be resorted to. Where the peritonitis is of a high grade, where repeated doses of opium are demanded to relieve the pain, and where from the severity of the symptoms it is probable that a fluid beginning to be purulent is present, the case should at once be placed in the hands of a surgeon, as the question of laparotomy will then have to be decided.

I find in my notes the record of a case of general peritonitis :

An infant (Case 447), nineteen months old, previously apparently healthy, was attacked with vomiting and diarrhoea. On the following day the face was pale, the alæ nasi were working slightly, the respirations were 36, and the temperature was 39.4° C. (103° F.). The respirations gradually increased to 74, and the temperature rose to 40.3° C. (104.6° F.). The abdomen became very much distended and tender, and the face pinched and anxious. On the evening of the second day from the onset of the disease the temperature rose to 41.1° C. (106° F.), the infant became very restless, the pupils were contracted, and death took place a few hours later.

The autopsy was made by Dr. W. F. Whitney.

The heart and lungs were normal.

The spleen was enlarged, and was covered with a fibrinous exudation.

The kidneys were pale, and normal in size.

The liver was covered with flakes of recent lymph, and on section showed the acini to be red and their periphery yellowish and opaque. The mesenteric lymph-glands were slightly enlarged, and the smaller ones were translucent on section and presented evidence of hyperplasia. A small pocket of the larger glands was found to have become cheesy in the central portions, and in two of these the process had extended through the substance of the gland and had broken through its peritoneal covering. About these points of rupture there was a small zone of reactive inflammation.

**PATHOLOGICAL DIAGNOSIS.**—Acute general peritonitis, which, from an absence of any other source, must be considered to have been caused by the rupture of the cheesy, degenerated mesenteric glands.

In this case the high temperature and the distended abdomen rendered the diagnosis comparatively clear. The case is important on account of the cause, for there is seldom any noticeable enlargement of the mesenteric glands under the age of three years, and these glands seldom soften, but either retrograde or harden from calcification.

**Chronic Peritonitis.**—When acute peritonitis is localized in one portion of the intestine it may become chronic and form fibrous adhesions, but in the majority of cases chronic peritonitis, especially when general, is of *tubercular* origin.

*Tubercular Peritonitis.*—The original source of the tubercular process is often obscure. It may be primary in the peritoncum, but is more likely to be secondary to tubercular mesenteric glands.



**PATHOLOGY.**—The process consists in the formation of miliary tubercles on the peritoneal surface, which give rise to opaque cheesy thickening, often nodular, with firm adhesions of the adjacent surfaces. An exudation into the peritoneal cavity is usually present, the quantity generally being considerable and the quality fibrino-purulent.

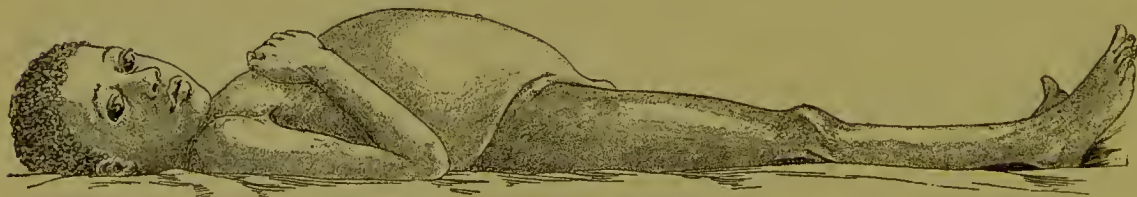
**SYMPTOMS.**—The initial symptoms of tubercular peritonitis are usually ill defined. There is a gradual loss of appetite and flesh, with occasional abdominal pain, which, as a rule, is not of a severe character. Attacks of diarrhoea are common, and are apt to be paroxysmal. The temperature is at times raised, especially in the latter part of the day. After these general symptoms have lasted for a number of weeks, the abdomen is noticed to be distended. A physical examination may show that there is nothing abnormal in the thorax, and that the morbid condition is confined entirely to the abdomen. At first the abdomen is resonant on percussion, but later may be dull, owing to masses of tubercle or to the presence of fluid. There is seldom any tenderness noticed on examining the abdomen.

**DIAGNOSIS.**—In a typical case, where the symptoms which I have just mentioned are present, the diagnosis is not difficult. Occasionally, however, there are no definite signs by which a diagnosis can be made, the only tangible sign being a seeming abdominal tumor, the resemblance of which to other abdominal tumors is so close that the diagnosis can be made only by laparotomy. You must nevertheless remember that most doubtful cases of abdominal tumors in children are tubercular.

**PROGNOSIS.**—When untreated, the prognosis of tubercular peritonitis is very variable. In some cases the disease after a number of months retrogrades, and the patient recovers. In most instances, however, the child becomes more and more wasted, the fever becomes more pronounced, the diarrhoea continues, the emaciation becomes extreme, and the child dies, usually of exhaustion. The surgical treatment of the disease has made the prognosis much more favorable.

#### CASE 448.

##### I.



Tubercular peritonitis. Male, 9 years old.

**TREATMENT.**—The treatment of tubercular peritonitis is essentially surgical, especially where there is ascites of any amount. In some cases, opening the abdomen and evacuating the fluid will not only give relief but will produce a permanent cure. In my experience at the Boston Children's Hospital, this procedure is often followed by complete arrest of the disease.

I have some cases of tubercular peritonitis here in the wards to show you.

This colored boy (Case 448, I., page 922) is nine years old.

His father died of phthisis. He has never been strong, but has had no acute illnesses. Three weeks ago he began to have diarrhoea, and soon after enlargement of the abdomen. There was no pain, vomiting, nor cough. He has lost greatly in weight. You see that he is emaciated. He has a temperature of  $38.3^{\circ}\text{C}$ . ( $101^{\circ}\text{F}$ ). His abdomen is much distended, and there is a distinct wave of fluctuation. Physical examination shows nothing else abnormal.

CASE 448.

II.



Tubercular peritonitis. Four months after operation.

(Subsequent history.) Laparotomy was performed by Dr. Bradford, and the fluid evacuated. Tubercle-bacilli were found in the peritoneal tissue. When seen six months later the wound had healed perfectly, and he was strong and well. This picture (II.) was taken four months after the operation.

CASE 449.

I.



Tubercular peritonitis. Male, 2 years old.

This next case (Case 449, I.), a boy, two years old, is especially interesting in regard to diagnosis.



He has not had general symptoms of serious import, but has lost slightly in weight, appetite, and strength. From time to time during the last six months he has complained of abdominal pain and tenderness. An examination of the abdomen shows a hardened, slightly irregular mass extending directly across the abdomen from one side to the other, 5 cm. (2 inches) above and the same distance below the umbilicus. It is not especially tender on pressure. Nothing else abnormal is detected about the child. As you see (II.), the line of percussion does not change when he is lying on his back, and there is no evidence of ascites.

## CASE 449.

## II.



Tubercular peritonitis.

(Subsequent history.) Laparotomy was performed by Dr. Lovett, and a mass of cheesy nodules matting together the intestine was found. An examination of a portion of this mass showed the presence of the bacillus tuberculosis. No fluid was present. The child recovered, but sufficient time has not elapsed since the operation to allow us to decide whether the disease will return.

## CASE 450.

## I.



Tubercular peritonitis. Male, 4 years old.

This boy (Case 450), four years old, was brought to the hospital some months ago with the extreme distention of the abdomen which you see represented in this picture (I.).

## CASE 450.

## II.



Tubercular peritonitis (after operation).

A physical examination showed nothing abnormal except in the abdomen, which was dull on percussion and showed fluctuation in every part. The child had gradually lost in weight, appetite, and strength.

Laparotomy was performed by Dr. Lovett, and a large amount of ascitic fluid evacuated. Tubercle-bacilli were present in the diseased peritoneum. The wound healed, but in the course of a few weeks the fluid reaccumulated, and laparotomy was again performed by Dr. Lovett. You see his condition now (Case 450, II., page 924), some weeks after the second operation. No fluid can be detected.

(Subsequent history.) There was no recurrence of the ascites, and the child recovered completely.

This boy (Case 451) is eleven years old

#### CASE 451.

##### I.



Tubercular peritonitis. Male, 11 years old. Four years after operation, showing scar under umbilicus.

He is, as you see, well and strong, and shows no symptoms of tubercular disease. You will notice the scar under the umbilicus, which marks the line of incision made when the laparotomy was performed.

#### CASE 451.

##### II.



Tubercular peritonitis.

He entered the hospital four years ago, and here is a picture (II.) of him which was taken at that time.



He had been perfectly well until four months before coming to the hospital, when he began to lose in weight and appetite and to show an increase in the size of his abdomen. Although he was not especially emaciated, he had lost in flesh and was pale. The circumference of the abdomen was 76.4 cm. (30 inches). On physical examination, nothing abnormal was detected in any of the other organs.

Laparotomy was performed by Dr. Bradford, and a large amount of serous fluid of a dark yellow color was removed. The peritoneum was found to be thickly studded with minute tubercles, and tubercle-bacilli were demonstrated. The peritoneal cavity was irrigated and drained.

For some months before the boy was attacked with tubercular peritonitis he had been drinking the milk of a tuberculous cow.

## DIVISION XV.

### DISEASES OF THE KIDNEYS, BLADDER, AND GENITAL ORGANS.

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#### LECTURE XLVII.

**KIDNEYS.**—Diseases of the kidneys may be congenital or acquired.

**CONGENITAL DISEASES.**—The congenital abnormalities, such as congenital cystic kidney, absence of one kidney, hypertrophy of the remaining kidney where one is absent, anomalous shapes of the kidney, and malpositions of the ureters, are important, but are so closely connected with purely surgical questions that they need merely be referred to in a medical lecture. The lobulated kidney, which I have already described (page 44, Fig. 9) as a normal condition in intra-uterine life, may to a greater or less degree continue into infancy and childhood, but has no pathological significance. Movable kidneys are rare in early life, but have been reported.

**ACQUIRED DISEASES.**—Renal disease as a primary affection in infancy and childhood has been considered rare, but this view has been modified by later bacteriological investigations, which have shown that nephritis is not uncommon in cases of general infection. Secondary renal lesions are comparatively common.

Renal diseases, with the exception of the nephritis following scarlet fever, have not been satisfactorily studied in children. A series of systematic examinations of the urine, in connection with later post-mortem examinations of the kidneys in the same cases, sufficiently extended to give us data for a precise diagnosis in an especial case, has not yet been made. Owing to the variation in the symptoms, the diagnosis of renal disease in the child must for the present depend upon the systematic and routine examination of the urine.

The diseases of the kidney in infancy and childhood are not so varied as in adults. They are chiefly represented by active hyperæmia (acute parenchymatous degeneration) and the nephritis following scarlet fever, which I have already fully described in my lecture on scarlet fever.

**PHYSIOLOGICAL ALBUMINURIA.**—Before speaking of the diseases of the kidney I shall describe a condition which is usually called physiological albuminuria.



This condition is not infrequent, and may occur at any period of infancy and childhood, but is most common between the fifth year and puberty. The amount of albumin present is, as a rule, less than one-twelfth per cent. It is not present in every micturition, and in many cases seems to depend upon over-exercise or a highly nitrogenous diet. The albumin is rarely present in the urine which is passed in the morning immediately after rising, and this is an important point in differentiating physiological albuminuria from periodic albuminuria due to pathological causes, such as uric acid. The children who have this physiological albuminuria often seem to be in good health, but sometimes they are rather delicate. The diagnosis can be made only by repeated examinations of the urine passed at different times in the day, and by observing the effect of exercise and diet upon it. The presence of blood-corpuscles or abnormal elements in any amount from the kidney shows that there is a pathological condition. An occasional hyaline cast and albumin as high as one-fourth per cent. for short intervals may be present. The albumin often disappears for a time and returns again. Children between the ages of three and seven years excrete nearly double the quantity of urine and of urea for each kilogramme of their weight that adults do. The amount of urea excreted in children between the ages of three and seven years is 0.973 gramme for each kilogramme of their weight. This fact is to be borne in mind in estimating the quantity of urea passed in cases of nephritis, because otherwise the kidneys might appear to be excreting a normal amount of urea and yet the amount be abnormally small for the age.

The prognosis in these cases of physiological albuminuria is good, and, so far as I know, no cases have been reported in which the condition terminated in nephritis.

The treatment of this condition is to regulate the diet, exercise, and general hygiene carefully. If the children are anæmic, iron is indicated.

GENERAL PATHOLOGY AND ETIOLOGY.—According to Councilman, to whom I am indebted for much information on this subject, the acquired diseases of the kidney in childhood show considerable differences from the renal diseases of the adult. In childhood there is a greater liability to those acute affections, such as scarlet fever, measles, and diphtheria, in the course of which nephritis is apt to appear. Children under the age of fifteen years are less subject to many pathological conditions, such as disorders of the circulation, which in the adult frequently lead to chronic lesions of the kidney. Children do not usually have those disorders of the circulation which result in granular kidney, for lesions of the arteries, especially the condition known as arterio-sclerosis, do not commonly occur in childhood. While it is true that typical examples of the small granular kidney are sometimes met with in children, these lesions of the kidney are primary, and the lesions of the circulatory system are secondary and dependent on the renal lesions. A part of the chronic diseases of the kidney in the adult is without doubt to be referred to the continuous action on the kidney of

slight pathological conditions, an action from which the child's age protects it. One pathological lesion not perfectly recovered from, moreover, makes the kidney more prone to disease, and a greater effect will be produced a second time by the same cause, and chronic disease will result. In the kidney of the adult, with the advance of years there is a gradual decline in the power of regeneration, and slight troubles are not readily recovered from. The kidney of the child, on the other hand, is an organ which possesses great power of growth and regeneration. For this reason a condition which in the adult organ is either not recovered from at all, or lays the foundation for chronic disease, will in childhood result in complete recovery. Again, the child is not exposed to certain conditions which are productive of chronic lesions, or which may lay the foundation for them. Among these may be mentioned alcoholism and excesses of various sorts. Many cases of nephritis in the adult are to be referred to causes acting not through the blood, but through the urinary tract. The child, on the other hand, is not exposed to the dangers arising from hydronephrosis and pyelonephritis, except to a very limited degree. Although the causes of disease are less numerous and less common in children than in adults, yet when the same etiological factor is present the same morbid condition is produced in the kidney. The various cachectic conditions will lead to amyloid infiltration in the child as they do in the adult, and amyloid infiltration of the kidney makes up by far the larger part of the chronic cases of albuminuria in children. We may also meet with certain chronic lesions in the child's kidney, such as are seen in tuberculosis, and these may lead to albuminuria and nephritis.

The acute diseases of the kidney, as a rule, either tend to recovery or are in themselves fatal; so that only a small number of chronic diseases are met with which result from the acute diseases. These are not to be referred to the continuous action of the poison of the acute disease, but to the effect on the kidney of the lesions produced by the acute process. An example of this is the condition of chronic nephritis after scarlet fever, where the acute lesions gradually pass into the chronic. These chronic lesions are to be attributed to the disorders in the circulation of the organ brought about by the destruction of the glomeruli.

**GENERAL SYMPTOMATOLOGY.**—The general symptoms connected with the various forms of nephritis are so similar that it will be less confusing to mention first the common symptoms which may occur in any of the forms of nephritis, and then to describe the etiology, pathology, and urinary examination of the different forms.

One of the most common symptoms in nephritis is œdema, which occurs frequently in acute nephritis and in chronic parenchymatous nephritis. The œdema generally appears first in the eyelids, and then in the hands and feet. There may be general anasarca. Not infrequently, however, œdema is absent or not marked. Vomiting is not infrequent in the beginning of the disease, and in some cases is, perhaps, due to the heightened temperature. It may



occur later in the disease as a symptom of uræmic poisoning. In such cases there is marked diminution in the amount of the urine, or even suppression. A peculiar dull white color of the skin is not uncommonly seen in chronic parenchymatous nephritis, and is quite striking. In acute nephritis fever is often present to a greater or less extent, but is a variable symptom. Lack of appetite, and weakness, are common in both acute and chronic nephritis. Headache is a variable symptom. It is a common symptom of uræmia, and sometimes the only one. Amaurosis may occur as the result of albuminuric retinitis, or it may be a functional symptom of the uræmic poisoning and disappear later if the patient recovers. Hypertrophy of the left ventricle is apt to occur in interstitial and chronic parenchymatous nephritis. Both diseases are, however, very uncommon in childhood. In acute nephritis following scarlet fever dilatation and moderate hypertrophy of the left ventricle are not uncommon. Transudation into the serous cavities has been reported in a number of cases, as has also œdema of the larynx.

Before I mention the details of the urinary analyses in the various diseases, you should understand that in all cases of nephritis the amount of urea should be carefully estimated from time to time, as a decrease in the urea always shows a pathological condition, and a return to the normal amount is usually indicative of recovery unless there is a complication with some other disease. Any interference with metabolism, whether in the liver or in the lung, may diminish the amount of urea in the urine. In children during convalescence from acute nephritis the urea returns to or exceeds the normal amount, while in chronic nephritis it is always diminished, as it is in adults. A sudden and excessive diminution of the urea in acute nephritis is suggestive of uræmia. In acute and chronic nephritis the chlorides are diminished when an effusion such as ascites is increasing, and gradually return to the normal amount as the effusion is absorbed.

**Active Hyperæmia** (Catarrhal Nephritis. Acute Parenchymatous Degeneration).—**ETIOLOGY.**—An active hyperæmia of the kidney may arise in the course of various acute infectious diseases. It may also be caused by an excess of uric acid, and by such irritating drugs as turpentine, cantharides, and arsenic. When the action of these causes is very intense, an acute nephritis may result.

**PATHOLOGY.**—The pathological conditions resulting from active hyperæmia of the kidney are a gradual degeneration and desquamation of the renal epithelium, and an injection of the blood-vessels. There is also to some extent an infiltration of round cells. The process seems to affect chiefly the epithelium of the tubules.

**SYMPTOMS.**—Unless the hyperæmia is very pronounced, there are, as a rule, no general symptoms, though œdema and other symptoms may rarely be present, as in acute parenchymatous nephritis.

**DIAGNOSIS.**—The diagnosis is made by the examination of the urine. The urine is clear, and its color is often normal. The amount is diminished. The specific gravity is higher than normal. There is a slight sediment, with

a trace of albumin, perhaps one-eighth per cent., or at times a little more. Microscopic examination shows the presence of renal epithelium and blood-corpuscles; the latter, however, not in sufficient number to color the urine. There are also leucocytes, and hyaline and fine granular casts, with an occasional epithelial cast and blood cast; the last three varieties, however, are not very numerous.

**PROGNOSIS.**—The prognosis in active hyperæmia of the kidney is good, and the pathological condition usually disappears when its cause has been removed.

**TREATMENT.**—The child should be placed on a diet exclusively of milk, so as to avoid any further irritation of the kidneys, and should be made to drink a great deal of water. It should be kept quiet, and its general hygiene should be carefully regulated.

I have here a case (Case 452) which is probably one of active hyperæmia. This boy, aged five years, had varicella when he was six months old, and measles when he was one year old. He had no other diseases until three weeks ago, when, without any apparent cause, he is said to have had a convulsion and to have vomited. He has never had any œdema, and a general physical examination shows nothing abnormal. An examination of the urine shows it to be high-colored and cloudy, to have a specific gravity of 1016, a large trace of albumin, and a yellowish-brown sediment consisting of amorphous urates. A microscopic examination shows the presence of uric acid crystals, hyaline, granular, and epithelial casts, and a few leucocytes. The total amount of urine passed in the twenty-four hours is from 360 to 450 c.c. (from 12 to 15 ounces). Heating the urine causes the high color, cloudiness, and brown sediment to disappear.

(Subsequent history.) Three weeks later there was only a slight trace of albumin in the urine, which was of a normal color, had a specific gravity of 1020, and contained a few hyaline and granular casts. A few weeks afterwards the urine was found to be normal. No other abnormal symptoms occurred during the whole course of the disease.

**Passive Hyperæmia.**—In addition to the active hyperæmia which I have just described, a *chronic passive hyperæmia* may occur, dependent upon diminished arterial or increased venous pressure. This condition occurs in chronic cardiac disease with disturbance of compensation, in chronic pulmonary disease, and where there is mechanical hinderance to the venous circulation, as from the presence of abdominal tumors.

**SYMPTOMS.**—The symptoms which occur in the course of passive hyperæmia are not referable to the kidney, but depend upon the disease which causes the hyperæmia. The urine in this condition is high-colored and often considerably diminished in amount. It has a high specific gravity, and often a heavy sediment of amorphous urates. There is a slight trace of albumin, usually under one-eighth per cent. Microscopic examination shows a few hyaline casts with renal cells adherent, and an occasional blood-corpuscle. There are, however, very few of these elements in the sediment. The peculiarity of the urine in passive hyperæmia is that it varies. If the heart becomes stronger, the urine is passed in larger quantities, is not so highly colored, and contains a smaller amount of albumin.

**PROGNOSIS.**—The prognosis in cases of passive hyperæmia of the kidney depends upon the cause of the condition.



**TREATMENT.**—The treatment is to be directed to the cause or causes of the congestion.

**Acute Nephritis.**—**ETIOLOGY.**—The most common cause of acute nephritis is scarlet fever. Other diseases in the course of which it may arise are diphtheria, measles, varicella, crysipelas, typhoid fever, malaria, pertussis, and pneumonia. With the exception of its occurrence in scarlet fever, diphtheria, and measles, the disease is not frequent. Cases have been reported where it has arisen in the course of extensive affections of the skin, such as eczema. It also occurs after the application of drugs to the skin, and from the internal administration of such irritating drugs as cantharides, turpentine, salicylic acid, and arsenic. Cases of primary nephritis have been reported where no cause could be found. Although it is difficult to estimate with certainty the importance of cold as a causative factor in the etiology of acute nephritis, and although it has been denied that cold can produce this condition, yet numerous cases have followed exposure to wet and cold. Many of these primary cases, however, were probably due to micro-organisms, as the disease has been not infrequently observed in connection with general septicæmia.

**PATHOLOGY.**—I have already fully described the pathology of the acute nephritis which follows scarlet fever. In the nephritis arising from the other causes which I have just mentioned, the pathological changes differ chiefly in the degree in which the different portions of the kidney are affected. The process appears to be a mixed one, but some portions of the kidney are more involved than others.

**SYMPTOMS.**—The symptoms of acute nephritis are such as I have already described in my lecture on scarlet fever. In general, the symptoms arising in cases due to other causes than scarlet fever are the same, but less severe than those which I have described in connection with that disease. The amount of albumin and the quantity of the urine depend chiefly upon the degree to which the glomeruli are affected. The number of casts and epithelial cells depends chiefly upon the degree of the involvement of the tubules. The interstitial changes can scarcely be determined by the urine. It is well to bear in mind that the urine may vary from day to day in any affection of the kidneys. In one type of the ordinary diffuse nephritis the urine presents the following changes. The color varies from red to brownish-red, according to the quantity and freshness of the blood which it contains. The specific gravity is high. The amount is markedly diminished, and there may even be anuria. There is a heavy dark-red sediment, with a large amount of albumin, usually more than one-quarter and often one-half per cent. Microscopic examination shows a large quantity of blood, numerous renal cells, leucocytes, a large number of casts, epithelial, blood, and fibrinous, also both fine and coarse granular casts and a fine detritus. All these elements are stained yellow or brown by the blood pigment. As the process advances towards recovery there are usually found, in a few days, more abnormal blood-corpuscles showing themselves in the form of

pale rings. There are more granular casts and detritus, and fewer epithelial and blood casts. Fatty elements, such as fatty renal cells and free fat, begin to appear. There are also more hyaline casts, usually with a few cells and blood adherent to them. Still later, there is a preponderance of hyaline casts, with fewer epithelial cells and blood-globules. During this time the amount of urine increases, until during the convalescence it finally rises above the normal amount. The color changes to smoky, and finally becomes pale. The specific gravity diminishes. The albumin diminishes to a trace, but this trace may persist for a long time. The elements in the sediment become fewer. Acute exacerbations are not uncommon.

**PROGNOSIS.**—In general the prognosis is good. The disease rarely becomes chronic. Death may occur in the beginning from the severity of the disease, or later from uræmic poisoning. Some cases end fatally from some intercurrent disease, such as pneumonia, or from a nephritis occurring in the course of a general septicæmia. The majority of the cases, however, recover after from four to eight weeks, although a trace of albumin and a few hyaline casts may persist for several months, the child in other respects being quite well.

**TREATMENT.**—The treatment of acute nephritis is the same as that which I have already described in the nephritis following scarlet fever.

**Chronic Parenchymatous Nephritis.**—**ETIOLOGY.**—Chronic parenchymatous nephritis is not a common disease in childhood, and its etiology is still very obscure. Some cases have followed an attack of acute nephritis, and in these there has generally been an interval during which the urine has simply contained a trace of albumin and a few casts, the symptoms of a chronic affection of the kidney appearing later. Cases have also occurred in connection with long-continued suppurative processes in the bones, joints, or elsewhere, arising in the course of tuberculosis or syphilis. In these cases amyloid infiltration is also apt to occur. There are also instances where no cause whatever can be discovered.

**PATHOLOGY.**—The pathological condition is the same as in the adult.

**SYMPTOMS.**—The symptoms of chronic parenchymatous nephritis are insidious in the beginning and are prolonged. There are marked pallor, a tendency to œdema, and a transudation into the serous cavities. Cardiac hypertrophy, weakness, loss of appetite, headache, and at times vomiting and diarrhœa, are among the common symptoms. Retinal changes sometimes occur, and there is a tendency to intercurrent diseases, such as pneumonia and pleurisy. Uræmic intoxication may be expected later. The urine may be high or pale in color. It is diminished in amount, but not markedly so, as in acute nephritis. The sediment is usually heavy. The specific gravity is diminished. There is a large amount of albumin, often one-half per cent. or more. There are frequently amorphous urates in the sediment, which must be removed by heat before the microscopic examination is made. Microscopic examination shows a characteristic preponderance of fatty elements. There are fatty renal cells, free fat, fat in the casts,



and cells completely fatty. There are also compound granular cells, and granular, epithelial, and hyaline casts. There are often acute complications in the kidney, in which case the amount of urine becomes markedly diminished, and the sediment shows blood, blood casts, and epithelial casts in addition to the large number of fatty elements. When the disease is complicated with amyloid infiltration, the diagnosis of the latter can hardly be made from the urine.

**PROGNOSIS.**—The prognosis is not good. Some cases having the clinical symptoms of the disease have apparently recovered. Most cases, however, die from uræmic intoxication or from some intercurrent disease, such as pneumonia. There may be a remission in the symptoms for a time.

**TREATMENT.**—The treatment is to restrict the diet as far as possible to milk. Good hygienic surroundings, and as much rest as possible, are indicated. Diuretics may be used when the amount of urine is diminished. I have already described in my lecture on scarlet fever the best treatment with diuretics (page 563).

Here is a boy (Case 453), eleven years old, with nephritis which has lasted a year. The examination of the urine by Professor Wood shows the probability of a chronic parenchymatous nephritis with an acute exacerbation.

CASE 453.



Probable chronic parenchymatous nephritis with an acute exacerbation. Male, 11 years old. Relapse after being out of bed five days.

This child had pertussis when he was three years old, scarlet fever when he was four years old, and measles and pneumonia when he was five years old. He is reported to have remained well from that time until nine months ago, when, without any known cause, such as exposure to cold or sickness of any kind, his face and eyes began to be œdematous. This was followed by œdema of the legs and ankles, and was accompanied by dyspnoea. The urine was noticed to be nearly of the color of blood, and to be lessened in amount. He was kept in bed for six weeks, and is said not to have complained of any especial discomfort. During this attack his appetite remained fair. Since the beginning of the attack he has grown somewhat weak and become pale. Six weeks ago the paleness and œdema about the

eyes increased, and the urine became smoky again. This was followed by œdema of the ankles, feet, and legs, accompanied by dyspnœa. The bowels have been regular, and there has been no vomiting. He sleeps well. On entering the hospital his face looked pale and waxy. There was considerable œdema of the face, especially of the eyes. His tongue was slightly coated, and there was œdema of the ankles, feet, and legs. Nothing abnormal was found in the heart or lungs, and there was no evidence of ascites.

He was kept in bed and given a diet of milk. Under this treatment the œdema and anæmia disappeared rapidly, and in two weeks he was allowed to be dressed and about the ward. This was five days ago. Yesterday he again had œdema of the face, and was immediately put to bed. As you see him to-day, the œdema under the eyes is especially prominent. From 750 to 900 c.c. (25 to 30 ounces) of urine are passed in the twenty-four hours. An examination shows it to have a specific gravity of 1010, an acid reaction, to contain urica 4.75 grains to the ounce, to have the chlorides diminished, and to contain  $\frac{6}{10}$  per cent. of albumin, but no sugar. The sediment shows numerous hyaline casts of medium diameter, some of large diameter from the straight tubules, many coarse and fine granular casts, numerous fibrinous casts, and many casts with renal cells adherent; also epithelial casts and blood casts; an excess of renal epithelium, most of it granular or fatty; compound granular cells, a large amount of abnormal blood, free fat, and fatty casts. His temperature has varied from 36.6° to 37.2° C. (98° to 99° F.).

(Subsequent history.) After remaining in the hospital for two months, with temporary periods of improvement, he was discharged in about the same condition as when he entered.

Here is a girl (Case 454), nine years old, with nephritis.

#### CASE 454.

##### I.



Probable chronic parenchymatous nephritis with an acute exacerbation, Female, 9 years old. Second week of the disease.

This child had measles when she was two years old, scarlet fever when she was three years old, varicella when she was six years old, and pertussis when she was eight years old. She apparently recovered entirely from all these diseases, and was well until one week ago, when, without any apparent cause, her face and feet began to swell. She complained of no pain, and had no other symptoms. As you see her in bed, you will notice the marked and extensive œdema of the entire face, body, and limbs. You see that the œdema is pronounced under both eyes, but especially so under the right one. There is great pallor of the skin, and the feet and hands are much swollen. Nothing abnormal has been detected



in the heart or lungs. There is no ascites. She has no headache, and does not complain of any discomfort.

An examination of the urine shows the color to be pale, the reaction acid, the specific gravity 1012, the sediment moderate; it contains albumin  $\frac{1}{4}+$  per cent., and no sugar; the sediment contains considerable abnormal blood, some free fat, and a number of hyaline and fine granular casts of medium and small diameter, many of them short and with oil-globules adherent. There are some fatty renal epithelium, leucocytes, casts with renal epithelium, and hyaline casts with a few renal cells adherent. There are also several fatty casts. The casts are not very numerous.

She is being treated by absolute rest in bed, bitartrate of potassium, digitalis, and a diet of milk, as I have recommended in my treatment of the nephritis following scarlet fever (page 545).

I show this child because she illustrates the appearance of a case of marked nephritis, with its excessive universal œdema and peculiar pallor of the skin. The diagnosis of the exact lesion of the kidney in this case is, however, very uncertain, as the pathological processes in the kidney are not confined to any one part of the organ, and the urinary analysis is often for this reason unsatisfactory.

#### CASE 454.

##### II.



Probable chronic parenchymatous nephritis with an acute exacerbation. Female, 9 years old. (Ten days after treatment was begun.)

I have provisionally called it a case of probable chronic parenchymatous nephritis with an acute exacerbation. The presence of blood may be due to an acute exacerbation, but might also mark it as an acute nephritis involving chiefly the parenchyma of the organ, as shown by the predominance of cells. It is significant in this case as pointing towards a chronic process that the urine has always been pale, showing that blood in sufficient quantity to color the urine has not been present.

(Subsequent history.) In about a week the œdema rapidly diminished and the urine increased in amount. An analysis of the urine at this time showed that the color was pale, that it had a specific gravity of 1010, a trace of albumin, and a slight sediment, consisting of a small amount of blood, renal epithelium, and a few casts with blood. The total amount of urine passed in the twenty-four hours was 2010 c.c. (67 ounces).

This picture taken at this time (II., page 936) shows how the general œdema has passed away, and how the skin has lost the extreme pallor which it presented on the child's entrance into the hospital.

An examination of the urine three weeks later showed the color to be pale, the reaction acid, the specific gravity 1014, the albumin  $\frac{1}{4}+$  per cent. It contained hyaline and fine granular casts of small diameter, many with oil-globules and renal cells adherent; also free oil-globules, fatty and granular renal epithelium, some normal and abnormal blood, leucocytes, and squamous cells. The casts were not very numerous, and there was not much change from what was found in the urine three weeks previously. At this time the urine again became scanty, and the œdema and pallor returned, but she did not complain of any discomfort. An examination of the urine eight weeks later showed it to be pale and cloudy, the reaction acid, the specific gravity 1018, and that it contained considerable sediment, and albumin  $\frac{1}{4}+$  per cent. The sediment consisted chiefly of hyaline casts of medium and small diameter, many of them having renal cells and fat adherent. There were also a few finely granular casts, considerable abnormal blood, free fat, fatty renal cells, epithelium, leucocytes, and occasionally blood, epithelial, and fatty casts.

The diagnosis cannot be positively established until the case shall have been under observation for a much longer period.

**Chronic Interstitial Nephritis.**—Chronic interstitial nephritis is so exceedingly rare in childhood that very little need be said concerning it. A few congenital cases have been reported.

**ETIOLOGY.**—The etiology is obscure. In some cases it seems to have followed a chronic parenchymatous nephritis. In others no cause could be found.

**PATHOLOGY.**—The pathology is the same as in adults.

**SYMPTOMS AND DIAGNOSIS.**—The diagnosis can scarcely be made from the symptoms. The disease is progressive and slow, with no characteristic symptoms. Cases have been reported in which there were general symptoms of headache, weakness, dyspnoea, palpitation, and disturbance of vision. Hypertrophy of the left ventricle occurs as a constant lesion. There is little tendency to anasarca; retinitis may be present. Baginsky refers to the lack of development of the children in these cases, and this condition was noticed in a case of this disease which occurred at the Boston Children's Hospital.

This child (Case 455), a girl, twelve years old, showed the development of a child of about seven years. The only symptom until she died of uræmic poisoning was persistent headache. The post-mortem examination showed marked interstitial nephritis, but it was not possible to determine whether it was primary or not, and no previous history could be obtained.

In chronic interstitial nephritis the amount of urine passed in the twenty-four hours is increased. It has a low specific gravity, a very slight sediment, and a trace of albumin. The microscopic examination shows a few hyaline and finely granular casts and occasional renal cells. Sometimes towards the end of the disease highly refractive homogeneous casts resembling wax appear in the urine. At this time the amount of urine may be somewhat diminished, but the specific gravity does not rise, as the excretion of urica is interfered with.

**PROGNOSIS.**—The prognosis is very unfavorable. The children usually



die of cerebral hemorrhage or of some intercurrent disease, the fatal result occurring in from three to four years.

**TREATMENT.**—The treatment is symptomatic.

**Amyloid Infiltration.**—In connection with amyloid changes in other organs, especially the liver, spleen, and intestine, amyloid infiltration may occur in the kidney.

**ETIOLOGY AND PATHOLOGY.**—It occurs at times in connection with chronic suppurative processes in the bones or elsewhere, and also in tuberculosis, syphilis, and chronic wasting diseases. It is not, however, especially common in early life.

**SYMPTOMS.**—The symptoms are not referable to the kidney. The presence of amyloid changes in the liver and spleen, shown clinically by enlargement and by the examination of the urine, are the signs by which the diagnosis is made. The urine is usually passed in large quantity when the amyloid changes are advanced. The specific gravity is low, and albumin is present. When the amount of urine is not much increased, as may happen temporarily, the albumin occurs in large amount. Microscopic examination shows no characteristic sediment; but when, as may often happen, the disease is combined with chronic nephritis, the sediment will show evidence of this latter disease.

**PROGNOSIS.**—On account of the usual causes of this condition, the prognosis is unfavorable.

**TREATMENT.**—The treatment is symptomatic.

**Pyelitis and Pyelo-Nephritis.**—**ETIOLOGY.**—Pyelitis and pyelo-nephritis may be due to an extension upward along the genito-urinary tract of an infection caused by catheters, gonorrhœa, or cystitis. Cases due to these causes, however, are uncommon in comparison with those which result from the excretion of uric acid by the kidney or from pelvic calculi. The disease may also be caused by tuberculosis of the kidney and by malignant growths.

**PATHOLOGY.**—The pathology of this disease varies with the cause. After the pyelitis has lasted for a time the kidney is affected in almost every case, and pyelo-nephritis results.

**SYMPTOMS.**—In an acute attack of the disease, as when it is caused by uric acid or a calculus, there are often pain and fever. Typical attacks of renal colic, with vomiting, pain, and fever, may occur. If the condition be due to tubercles, malignant growths, or abscess of the kidney, there will be more or less cachexia and emaciation, and there may be local pain and tenderness.

The diagnosis is to be made from the examination of the urine. The urine contains pus, which gives it a cloudy appearance, and the sediment is heavy. The color varies: it may be red if there is considerable hemorrhage. The urine contains albumin, which varies from a trace to a considerable amount, according to the amount of blood or pus and the presence or absence of a concurrent affection of the kidney. The microscopic examination shows

sometimes the whole field to be filled with pus-corpuscles, at other times the pus to be in clumps; there are also present small round cells with single nuclei, from the pelvis or from the kidney, and more or less blood. The diagnostic cell of pyelitis is the "caudate cell," which is a small cell about the size of a renal cell, having a single nucleus and a tail. If the kidney is affected there are casts of various kinds, hyaline, granular, epithelial, and blood. The casts may not be easily seen if the field is filled with pus. The presence of tubercle-bacilli in the sediment, shown by appropriate methods of staining, establishes the diagnosis of tuberculosis. In the freshly passed urine, uric acid is often present in the sediment in the form of irregular spiculated crystals. These may suggest the probable cause of the pyelitis.

**PROGNOSIS.**—The prognosis depends upon the cause. In malignant growths it is fatal. This is true to a greater or less degree where tubercle is the cause of the disease, as in almost every case it is present somewhere else in the body. When uric acid or a calculus is the cause, the prognosis is more favorable, and, as a rule, the outcome depends upon the fact whether the treatment is appropriate or not.

**TREATMENT.**—The uric acid should be treated by neutralizing the acidity of the urine, by placing the child upon a mild and unirritating diet, such as milk, and by making it drink freely of distilled water. Operative treatment is at times called for where a calculus is present.

**Malignant Growths and Enlargement.**—Tumors of the kidney are more common and more serious in the child than in the adult. The simple adenomata are probably equally common in both, but the child is much more liable to carcinomata and sarcomata than is the adult. Sarcomata are the most common in the first five years of life, and usually occur in one kidney.

**SYMPTOMS AND DIAGNOSIS.**—The diagnosis depends upon the recognition of a tumor of the kidney and the progressive emaciation and cachexia which arise. At times there is pain, but, as a rule, pain is absent. The urine sometimes gives evidence of a pyelo-nephritis; at other times hæmaturia and albuminuria occur at intervals, but generally late in the disease, at a time when the tumor can be felt through the abdominal wall. Some of the characteristics of a tumor of the kidney are that it is located in the hypogastric and lumbar regions, that it is deep-seated, and that it is not so commonly to be felt in the umbilical region as are tumors of the retro-peritoneal glands. The tumor is irregularly rounded, and usually does not have a well-marked border, such as is found in enlargement of the spleen and liver. In these cases of sarcomata of the kidney the health at first is often not much affected, but there are progressive emaciation and enlargement of the abdomen, commonly without pain.

**PROGNOSIS.**—The prognosis is very unfavorable, although temporary relief is often obtained by means of surgical interference.

**TREATMENT.**—The treatment is essentially operative.



**AFFECTIONS OF THE SUPRA-RENAL CAPSULES.**—The affection of the supra-renal capsules called *Addison's disease* has been met with in young children, but is exceedingly rare.

**HÆMATURIA AND HÆMOGLOBINURIA.**—Hæmaturia and hæmoglobinuria are, as a rule, easily recognized by the color of the urine if sufficient blood is present to color it. The color is red if it is due to fresh blood, or brownish red if due to blood-pigment which has been washed out of the corpuscles.

To determine the source and cause of the hemorrhage is often quite difficult. Hemorrhage from the bladder may be caused by a calculus, or by papillomatous growths, or may occur in cases of hæmophilia. When the blood comes from the bladder it is generally not uniformly diffused through the urine, and small clots are common. In addition to this there are symptoms of disturbance of the bladder, such as tenesmus and frequent and perhaps interrupted micturition. In hemorrhage from the kidney the blood is diffused through the urine. The color may be red or brownish red. The microscopic examinations show epithelium and casts from the kidney, and the elements are stained yellow and brown from longer contact with the blood. There are also normal blood-corpuscles, and others from which the hæmoglobin has been washed out, appearing as pale rings.

Hæmaturia may occur in hæmophilia and in purpura. Hæmaturia may also be a symptom of malignant growth in the kidney. It may be an early symptom occurring at intervals, but usually it appears at a later period, when the presence of a tumor can be detected by palpation. It may also be caused by uric acid.

In cases of hæmoglobinuria, notwithstanding the red or at times almost black color of the urine and the presence of albumin, there are no corpuscles to be found. Heller's test, which consists in adding hydrate of potassium to the urine and heating it, causes a precipitation of the phosphates, which carry down the blood-pigment mechanically as dark-red flocculi. A similar appearance may be given to the urine after the administration of senna and rhubarb. In such cases Heller's test would give the same results as if blood-pigment were present. The nitric acid test for albumin would, however, decolorize the urine, and the test for albumin would be negative. It is important to recognize the very dark urine resulting from carbolic acid poisoning, as it occasionally occurs after the external application of this drug. Under these circumstances the urine has a greenish tinge.

**ETIOLOGY.**—The etiology of paroxysmal hæmoglobinuria is as yet obscure. The child often appears to be in good health. The most frequent apparent cause is cold. Certain individuals have hæmoglobinuria whenever they are chilled, or wet their feet, or plunge into cold water. Some cases of hæmoglobinuria appear to be due to infection, as in scarlet fever, Winckel's disease, and malaria. Certain inorganic substances when taken by the mouth, especially chlorate of potassium, phosphorus, and arsenic, have produced hæmoglobinuria.

Some cases of hæmoglobinuria have severe symptoms at the time of the attack, such as chills, cold extremities, and a rapid, small pulse. Neither these symptoms nor the hæmoglobinuria last very long, as a rule. At times it is impossible to determine the cause of the hæmoglobinuria. A case which has lately come under my notice shows how difficult it is to determine the cause of this disease even when a post-mortem examination can be obtained.

A girl (Case 456), four years and eight months old, had for several weeks grown pale, lost in weight, and shown symptoms of indigestion. Later the urine was reduced in amount and was dark-colored. There was also slight œdema of the eyelids and feet. No other especial symptoms arose, and the child went out of the house as usual and seemed otherwise well. For three or four days before her death the pallor and œdema increased markedly, and the urine was lessened in amount and became still darker in color. About twenty-four hours later she became very dull, and on the following day was much blanched and almost unconscious, except that when she was aroused to be examined she would resist and scream. The urine showed the condition of hæmoglobinuria. The child died a few hours later.

The post-mortem examination, made by Professor Councilman, showed evidences of profound anæmia. The bone-marrow was red. There were hæmoglobinuria, fatty degeneration of the heart, liver, and kidneys, and hæmoglobin casts in the tubules of the kidney.

**CHYLURIA.**—Chyluria is a rare disease. Two forms are usually spoken of, the tropical and the non-tropical.

**ETIOLOGY.**—The tropical form is caused by a parasite, the *filaria sanguinis hominis*, a species of round-worm. This parasite is found in the blood, and at times in the urine, especially that passed towards night. The exact connection between the parasite and the chyluria has not yet been determined. In the non-tropical form the parasite has not been found. Cases have been reported where the parasite appeared in an individual residing in the tropics, and disappeared on his returning to a cold climate, although the chyluria continued. The chyle is supposed to get into the urine after it has left the kidney.

**SYMPTOMS.**—The symptoms of this disease are shown chiefly in the urine. The urine has a milky appearance, sometimes a sour odor, and tends to decompose rapidly. The reaction is slightly acid, or neutral. Microscopic examination shows the fluid to be filled with fine fat drops in suspension. The urine at times contains blood-corpuscles, and albumin is always present. The attacks are apt to be paroxysmal, lasting for days or weeks, then ceasing and again recurring. A fatty diet may or may not cause an increase in the chyluria. The individuals affected by the disease may have a healthy appearance. Coagula may at times be formed in the bladder and give rise to pain and difficult micturition.

**PROGNOSIS.**—The prognosis of chyluria is doubtful. It is a disease which lasts for a long time and may cause anæmia and emaciation from the loss of fat and albumin.

**TREATMENT.**—There is no treatment which is known to be beneficial.

**Hydronephrosis.**—**ETIOLOGY.**—Hydronephrosis may be congenital, in which case it may be due to constriction of the ureter. Both kidneys may



be affected, but usually only one is involved. When acquired it generally affects but one kidney, and may be caused by obstruction to the escape of urine either from above, as by an impacted calculus in the hilus of the kidney or in the urethra, or from below, by the pressure from a tumor or enlarged mesenteric glands. The effects are mechanical, and are due to the pressure of the retained fluid on the kidney, which leads to the gradual absorption of the kidney-substance. These tumors sometimes acquire a large size.

**SYMPTOMS.**—The main symptom of hydronephrosis is the presence of an abdominal tumor connected with the kidney. When the tumor has grown sufficiently large, fluctuation can be usually detected, and aspiration gives a fluid which ordinarily contains urea. Subjective symptoms may be absent. If only one kidney is affected, the other performs the function of both, and the general condition of the child may remain good.

**PROGNOSIS.**—The prognosis is doubtful. Cases have been operated upon with success both by aspiration and by removal of the tumor.

**TREATMENT.**—The treatment of this disease is essentially surgical.

**ACUTE CYSTITIS.**—Acute cystitis is not a common affection in infancy and childhood.

**ETIOLOGY.**—It may be caused by a vesical calculus, by irritants, such as turpentine, and also occasionally by the extension of infection through the genital tract.

**SYMPTOMS.**—The symptoms of acute cystitis in children do not differ from those which are met with in the adult. The chief symptom is frequent and painful micturition. This local symptom is usually accompanied by fever, which may be high, and by general symptoms of malaise, fretfulness, and crying from vesical pain. The urine is passed in small quantities, and, as a rule, is of a reddish color. The specific gravity is high. When freshly passed it is acid, but it quickly becomes alkaline; there is a heavy sediment, and it contains a trace of albumin. Microscopic examination shows chiefly pus in large quantities, squamous epithelium, and some blood. To establish the diagnosis it is necessary to obtain the urine by the catheter, or, in females, first to wash out the vagina thoroughly, as the epithelium of the vagina and that of the bladder are very similar.

**PROGNOSIS.**—The prognosis of acute cystitis is good after the removal of the cause.

**TREATMENT.**—The especial cause of the attack must be looked for, and removed if possible. The child should be kept perfectly quiet in bed, and should be made to drink a great deal of water. The diet should be of milk. Sedatives should be used freely.

**CHRONIC CYSTITIS.**—Chronic cystitis may be caused in children, as in adults, by a vesical calculus, by foreign bodies in the bladder, by tumors, by papillomata, and by tuberculosis. The nuclei of the calculi are generally composed of uric acid, upon which phosphates are precipitated in alkaline urine, and this deposition is favored by the accompanying catarrhal inflammation.

**SYMPTOMS.**—Micturition is frequent and at times painful. Later there may be a constant dribbling of urine, giving rise to an offensive ammoniacal odor and causing irritation about the genitals. Where there is a calculus in the bladder the stream is often suddenly interrupted during micturition and the pain is more severe. Prolapse of the rectum is not uncommon with stone. In addition to these local symptoms there are general symptoms of anæmia and loss of weight. The urine is ammoniacally alkaline, offensive in odor, and turbid, has a heavy ropy sediment, and contains a trace of albumin. The sediment should be examined as soon as possible after the urine is passed, because the ammonia which is produced from the urea disintegrates the cells. The examination will show a large quantity of pus, some blood, bladder-epithelium, and crystals of triple phosphate and urate of ammonium.

**PROGNOSIS.**—The prognosis of chronic cystitis depends upon the cause, upon the length of time during which the disease has persisted, and the presence or absence of a secondary affection of the kidney.

**TREATMENT.**—The urine should be diluted by giving distilled water in large amount. It may be rendered less irritating by such drugs as salol and buchu, and less alkaline by benzoate of sodium. Washing out the bladder is of use in many cases, and local applications may be made in tuberculosis of the organ. Operative treatment is indicated when a calculus is causing the disturbance.

At times it is exceedingly difficult to determine by the general symptoms whether a calculus is present in the bladder. I shall report to you a case which illustrates this difficulty.

A boy (Case 457), seven years old, began to have pain of a spasmodic character in the region of the bladder during micturition. In connection with the pain there would be a sudden stoppage of the flow of the urine and a bearing-down feeling in the rectum. These symptoms simulated those of a vesical calculus so closely as to render a differential diagnosis very difficult. The boy was of a nervous temperament, and was rather anæmic, but otherwise was well and strong. Nothing abnormal was detected about the prepuce or the rectum. The pain was so annoying and caused so much trouble that it was deemed advisable to have the bladder examined for stone. An examination was made by Dr. Bradford, and nothing abnormal was detected. After the bladder had been examined, a decided improvement took place, apparently connected with the passing of the stone, and the boy recovered entirely after remaining at home from school for a few weeks and having daily exercise in the open air.

**VULVO-VAGINITIS.**—Vulvo-vaginitis is a very common affection in little girls. It arises from a variety of irritations, one of which is the oxyuris vermicularis. In a very large number of cases the gonococcus of Neisser has been found in the purulent secretion. The gonococcus was found in all of six cases lately treated at the Boston Children's Hospital. The disease may also arise in children who are very much debilitated, and is met with at times in scarlet fever and in measles. Again, it is not infrequent in anæmic girls, in whom it occurs without any apparent cause.

**PATHOLOGY.**—The labia are reddened and are more or less swollen.



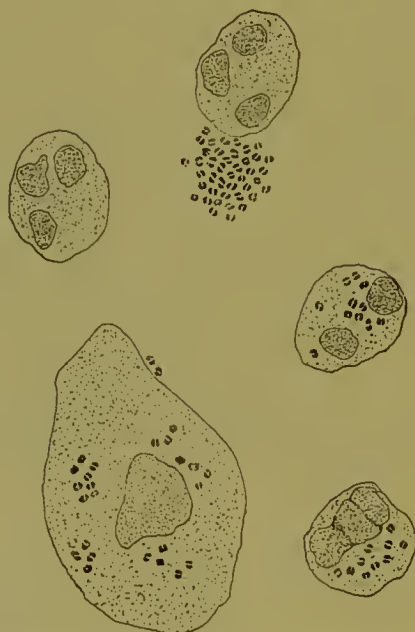
There is a thick, purulent discharge of a greenish-yellow color, usually offensive. At times there is more or less excoriation of the inner surfaces of the labia. The inguinal glands may be slightly enlarged and tender. The urethra is, as a rule, involved in the irritation, and is swollen and red.

**SYMPTOMS.**—There may be some fever in the early stages of vulvovaginitis. Smarting and burning are usually complained of, but at times the staining of the clothing first calls attention to the disease. The children commonly become pale if the disease persists for some time. Micturition is painful in some cases, and the disease is one of the many causes of dysuria. In many cases the children appear to be quite well, with the exception of the local condition.

**PROGNOSIS.**—The prognosis is good, but the disease is apt to be prolonged for several weeks or months. Complications may arise from the extension of the process into the urethra and the bladder, and cause additional symptoms referable to these parts.

**TREATMENT.**—Local applications to the vagina constitute the only satisfactory form of treatment. This is difficult in young children, but may be accomplished with a soft rubber catheter. Such solutions as boracic acid 4–100, corrosive sublimate 1–5000, or creolin 1–500, may be used. In some severe cases local applications of nitrate of silver 1 or 2 per cent. may be necessary. The labia should be kept separated by absorbent cotton, and the parts kept dry and covered with some mild dusting-powder. Absolute

FIG. 132.



Gonococci contained in pus-cells from male, 8 years old. Acute stage of inflammation.

cleanliness must be observed, to prevent infection of the eyes and of other persons. The parts should be protected with compresses held in place by a bandage, which should be worn all the time, and the compresses should be frequently changed and burned. The towels used for the patient should not be left lying about, and should be carefully disinfected. Tonic treatment is

sometimes indicated. The urine should be kept dilute, in order to avoid irritating the inflamed surfaces, and any complicating cystitis should be treated. During the active stage of the disease the child should be kept as quiet as possible, and on a diet of milk.

Where the vulvo-vaginitis is caused by the *oxyuris vermicularis*, especial care should be given to eradicating the parasite from the rectum. After this has been done, the vagina is readily freed from the parasite by using an injection of warm sweet oil, which is to be allowed to remain for three or four minutes, the vagina then being syringed out with warm water.

Gonorrhœa also may occur in boys.

This boy (Case 458), eight years old, came to the hospital yesterday complaining of pain on micturition and on walking. The prepuce was found to be very much swollen, and there was a discharge of pus from the urethra. An examination of the discharge by Dr. Mallory showed the presence of gonococci in the pus-cells. This specimen (Fig. 130, page 944), taken from this case, shows the morphology of the parasite as seen by means of a Leitz homogeneous oil immersion  $\frac{1}{12}$ , Leitz stand Oc. No. 3, tube closed.

The gonococci are ovoidal or biscuit-shaped, and usually occur in pairs, the flat sides being opposed to each other. It is characteristic of them that they are found within the pus-cells as well as on their surfaces and free in the fluid.

**ORCHITIS.**—Orchitis, or inflammation of the testis proper, may occur in childhood from direct injury, but it is a rare disease. When present it is commonly accompanied by hydrocele. The orchitis which so commonly follows mumps in the adult is less common in children.

**EPIDIDYMITIS.**—Besides being due to trauma, acute epididymitis may be caused by any irritation of the mucous membrane of the urethra. In this disease the whole scrotum is apt to be hot and tender, and the child is in great pain. The epididymis is much enlarged and exquisitely tender, and pushes the testis forward. The cord is often implicated, becoming enlarged and painful on pressure.

The treatment should be energetic, as, owing to the swelling of the tissues about the testicle, there may be so much pressure that the gland will be seriously damaged, although the subsequent atrophy may not declare itself for a considerable time. The child should be kept upon his back in bed, the bowels freed with a cathartic, and a series of hot poultices kept upon the scrotum. In all inflammations of the testis or epididymis the scrotum should be placed in such a position that the lower end of the testicle points upward.

**TUBERCULAR DISEASE OF THE TESTICLE.**—As compared with the frequency of its occurrence in adults, tubercular disease of the testicle is rare in infancy and childhood. When the disease is present the gland is considerably swollen and often nodular, but rarely very tender. As the disease progresses, adhesions may form with the tissues of the scrotum, and the degenerated material may be discharged through a fistulous tract.

General treatment is indicated if the disease is just starting, but if it has already destroyed the usefulness of the gland it is safer to operate



immediately and remove the focus of infection ; here, of course, we should be guided by the conditions elsewhere.

**TUMORS.**—In addition to tubercular disease of the testis, tumors may be found in infancy and early childhood. These may be congenital or acquired. The congenital tumors are very rare, and are usually of the dermoid variety. The most common of the acquired tumors are sarcomata, which are very malignant. The rapid growth and the large size of this variety usually render the diagnosis easy.

**PHIMOSIS.**—In early life there appears to be a physiological adhesion of the prepuce to the glans penis. As the child grows older these adhesions normally disappear. When the adhesion between the prepuce and the glans remains permanent and the prepuce is very tight, the condition gives rise to various symptoms. Thus the escape of the urine may be mechanically hindered, and the urine collecting behind the glans may give rise to irritation. Smegma is also apt to collect around the corona. In this way an inflammatory condition of the prepuce (posthitis) or of the glans (balanitis) may arise. As a result of this there is swelling, and micturition is painful and difficult. In addition to these local symptoms many secondary disturbances arise from the local reflex irritation. Among these are nervous phenomena of greater or less degree, such as convulsions. Phimosis may lead to enuresis and masturbation.

In all cases of phimosis local treatment is indicated, and may be by dilatation, incision, or circumcision.—the latter being the most radical and producing the best results for complete relief from the morbid condition. In all cases, even if the phimosis is very slight, mechanical interference should be persisted in until absolute cleanliness can be secured, for in this way only will entire relief from the local and reflex symptoms be obtained.

**ANURIA.**—I have already spoken of the forms of anuria which result from suppression of the urine in nephritis. Anuria may also occur in infants and in young children irrespective of any disease. The infant will not pass its water for perhaps twenty-four hours, apparently from no especial cause.

Hot applications over the bladder and making the child drink an increased amount of water will usually relieve this condition. It seldom calls for the use of the catheter, and serious results need not be apprehended.

**ENURESIS (Incontinence of Urine).**—Enuresis is a condition in which there is an involuntary discharge of the urine. It may be continuous or periodic. It may also be diurnal, nocturnal, or both. It is of very frequent occurrence in infancy and early childhood. It is a symptom rather than a disease, and in most cases is a true neurosis. During the first year of life the infant has not learned to assume control of the mechanism of micturition, but during the second year this control is usually attained at an earlier or a later period according to the individual.

**ETIOLOGY.**—The causes of enuresis may be organic or functional, the latter in all probability being very commonly of a reflex nature.

The organic causes comprise such malformations as small ureters, a small bladder, exstrophy of the bladder, and hypospadias. Enuresis may also be caused by central lesions of the brain and cord.

The prognosis and treatment of these organic cases of enuresis vary according to the conditions which cause them, and need not be considered here. In a large number of cases the children are of a highly nervous temperament, but enuresis is also often present in children who otherwise do not show any nervous symptoms. As has been stated by Rachford in an admirable paper on this subject, this condition may depend upon (1) irritable and unstable nerve-centres, (2) anæmia with malnutrition, and (3) reflex stimulation of certain nerve-centres in the lumbar cord. The longitudinal and circular muscular fibres of the bladder, which by their contraction empty the bladder, are innervated by sensory and motor nerves from the lumbar region of the cord, and the external sphincter in the prostatic portion of the urethra, which by its contraction prevents the escape of urine from the bladder, is also innervated by sensory and motor nerves from the lumbar cord. The researches of Von Zeissl show the manner in which reflex causes may act in starting or checking the flow of the urine. Thus, a reflex carried to the proper centre in the lumbar cord would, through the motor fibres of the erector nerve, contract the muscular coat of the bladder, and through the inhibitory fibres of the same nerve relax the sphincter vesicæ. In this manner the urine which is being expelled by the contracting bladder is allowed to pass without hinderance through the relaxed sphincter vesicæ. It is also to be remembered that the act of urination is in part under the control of the will. Admitting these anatomical and psychical facts, it is easily understood how the causes which produce enuresis may act in two ways: either directly on the centres in the lumbar cord, making them more irritable or unstable, and in that way increasing their reflex excitability, or indirectly through exaggerated reflex causes that affect both accelerator and inhibitor influences sent to the bladder. These influences may be psychic, originating in the brain, or may be the result of external irritation originating in or near the bladder itself.

There is also during childhood a lack of development of the centres of inhibitory reflex acts, and in this way the muscular fibres of the bladder, having no inhibitory restraint, are excited to action by even so slight a reflex cause as a small quantity of urine in the bladder. For this reason enuresis is a normal condition during infancy, and ceases when the child's inhibitory mechanism is more developed (Soltmann). The inhibitory influence of the will is in abeyance during deep slumber, and nocturnal incontinence is therefore more frequent than diurnal. In any diseases which are accompanied by anæmia and malnutrition the reflex irritability of the lumbar nerve-centres is much increased, and enuresis may result. Reflex enuresis may be caused by irritation in any portion of the genito-urinary tract, as by a vesical calculus, cystitis, vulvitis, phimosis, very acid urine, and over-filling of the bladder, as in diabetes, or by an irritation of some



neighboring part, such as may arise from a polypus or the oxyuris vermicularis in the rectum.

**SYMPTOMS.**—As a symptom, enuresis is simply the involuntary emptying of the bladder.

**PROGNOSIS.**—The prognosis of enuresis varies greatly, according to the cause and the individual. In a large number of cases the enuresis lasts for only a short time, but in some cases it may continue throughout childhood: almost invariably, however, it ceases between the twelfth and the fourteenth year. The cases in which enuresis does not disappear at puberty are nearly always in girls.

**TREATMENT.**—The treatment of this functional form of enuresis is often very unsatisfactory. According to my experience, in quite a number of cases the disease is intractable and is not affected by any treatment whatever, the individual finally recovering without treatment. After a careful examination has shown that no malformation or central nervous lesion is present, the urine should be examined, to determine if it is abnormally acid. When this is found to be the cause of the irritation, a rapid cure can be effected in some cases by simply diluting the urine. In females, especially when there is irritation around the meatus urinarius, local applications are of great service, and in some cases dilating the urethra will produce a permanent cure. Where phimosis is present, relief has been sometimes obtained by circumcision. The bowels should be regulated, and it is well to have the child pass its water just before going to sleep, and to rouse it in the middle of the night in order that it may empty its bladder. The foot of the bed should be raised, in order that the urine shall not irritate the neck of the bladder. There is no especial drug which in my experience can be relied upon in curing enuresis. Where the children are anæmic and debilitated, iron and nux vomica are indicated. Where there is excessive irritability of the nerve-centres, belladonna and atropine are at times efficient in relieving this condition; but in many cases they fail to produce beneficial results even when given in toxic doses. Faradism applied to the perineum, or to the base of the sacrum and to the symphysis pubis, is in some cases beneficial. There is, however, no routine treatment for enuresis. Each case should be studied closely, and in many instances when the especial cause of the condition has been found the enuresis can be relieved.

## DIVISION XVI.

### DISEASES OF THE LARYNX, TRACHEA, LUNGS, AND PLEURA.

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#### LECTURE XLVIII.

##### DISEASES OF THE LARYNX AND TRACHEA.

LARYNGOSPASMUS.—NEW GROWTHS.—FOREIGN BODIES.—ŒDEMA.—LARYNGITIS.

**LARYNX.**—The affections of the larynx which occur most commonly in infants and young children are neuroses, new growths, lesions produced by foreign bodies, œdema, and laryngitis.

**LARYNGOSPASMUS** (*Laryngismus Stridulus*).—The neurosis which especially affects the larynx in infancy and childhood is what I have already described under the name of laryngospasmus when speaking of reflex irritation of the larynx in my lecture on Nervous Diseases (page 747). I shall therefore merely refer you to what I said at that time concerning it.

**NEW GROWTHS.**—New growths in the larynx in infants and children are rare. They may be congenital, but these are very uncommon. They may be malignant, such as epitheliomata and sarcomata, or benign, such as fibromata, myxomata, and papillomata. Those of the former class are so rare that they need here only be referred to. Of the latter class the fibromata and myxomata are too rare to be more than mentioned. The papillomata, on the other hand, although rare, are the most common laryngeal growths in early life. They may produce such serious results that it is important to recognize them at once. They may be congenital. Their cause is not known. Papilloma of the larynx in young children is usually multiple.

The symptoms of this growth appear at about the first, second, or third year. The first symptom that is noticed is hoarseness. This hoarseness, instead of passing off in a few days, as is common where it arises from other affections of the larynx, continues and grows more marked. The next symptom is dyspnœa. This appears at intervals of a few months, or may not arise for some years after the first alteration of the voice. The dyspnœa first appears at night, when the child is asleep. In the daytime, when the



child is awake and running about, it may breathe freely. As the papillomata increase in size, the dyspnoea appears in the daytime also, especially when the child makes any exertion. When the child is awake and is quiet the breathing may not be noticeably affected, even after the growth has attained a large size. Cough may be present. Usually there is no pain or difficulty in swallowing. When a child presents these symptoms a careful laryngoscopic examination should be made at once, as in this way only can the diagnosis be verified.

The prognosis in these cases is bad unless the growths are removed.

The best treatment of multiple papillomata is to etherize the child and remove the growths through the mouth.

The difficulty of removal is in some cases so great that some of the most competent operators have preferred to postpone the operation until the child is older, or until the symptoms are so urgent that there is danger of suffocation. The child during this time must be kept under strict supervision, but local applications are not indicated. These growths, even when completely removed, have a tendency to recur.

**FOREIGN BODIES.**—Foreign bodies rarely lodge in the larynx, but this accident occurs more commonly in children than in adults, as children are apt to put articles of every description into their mouths.

The symptoms which indicate the presence of a foreign body in the larynx are an attack of sudden suffocation and a change in the sound of the voice in a child who has previously shown no signs of obstruction and no symptoms of laryngeal disease.

The accident is one which is so serious that the child should be placed at once in the hands of a laryngologist. The larynx should be examined with the laryngoscope, and the foreign body removed, if possible, with the forceps. Great care should be taken not to push the foreign body into the trachea, as tracheotomy would then be necessary. For the same reason it is inadvisable to introduce the finger blindly into the larynx, or to do anything which may cause a sudden inspiration.

**ŒDEMA.**—Œdema of the larynx is not a common condition in early life. It may arise from a number of causes, and is secondary to some disease elsewhere or to some local irritation. It occurs as a rare complication in nephritis and in the acute exanthemata. It may arise from irritation produced by local lesions, such as ulcerations, from foreign bodies, from inhalations of hot vapors, from the swallowing of corrosive liquids, and also as the result of any acute inflammation, such as erysipelas.

The diagnosis, as a rule, must be verified by a laryngoscopic examination.

The treatment is that of the disease or local irritation which is causing the œdema. The local application of cold, and, if necessary, scarification of the œdematous tissue, are indicated. If the attack is pronounced and suffocation is imminent, you should be in readiness to perform tracheotomy or intubation.

**LARYNGITIS.**—The most common inflammatory lesions of the larynx which occur in early life are (1) catarrhal (false croup, croup) and (2) pseudo-membranous (membranous croup).

**Acute Catarrhal Laryngitis.**—The pathological condition which is present in the acute form of laryngitis is a redness or hyperæmia of the laryngeal mucous membrane, accompanied by more or less swelling and serous exudation. The cause of acute catarrhal laryngitis is often a simple extension of a catarrhal condition of the nose and pharynx to the larynx. More rarely a catarrhal condition of the bronchi and trachea may extend upward and involve the larynx. At times the condition appears to be the result of atmospheric changes and undue exposure to dampness and cold. The lumen of the larynx in infancy and in early childhood is so small that even a moderate swelling of the laryngeal mucous membrane may produce sufficient stenosis to give rise to marked obstructive symptoms.

**SYMPTOMS.**—The symptoms of acute laryngitis are a heightened temperature,  $38.3^{\circ}$ ,  $38.8^{\circ}$ ,  $39.4^{\circ}$  C. ( $101^{\circ}$ ,  $102^{\circ}$ ,  $103^{\circ}$  F.), and even higher, hoarseness, and cough. These symptoms, occurring in connection with a preceding rhinitis or pharyngitis, or arising from a primary inflammation of the larynx, may continue for a number of days without any more serious manifestations, and if the child is kept in an equable temperature the attack may pass off within a week. In some cases, however, another set of symptoms may appear after the primary manifestations have lasted for a variable period. The child may have been as well as usual during the day, and may have been playing about. Towards the latter part of the day its voice may have become hoarser, but otherwise no especial symptoms may have arisen. The child, after being restless for a time, suddenly awakes, and springs up in bed frightened, often clutching at its throat as if it had a sensation of suffocation. The cough, which during the day was hoarse and somewhat metallic, is now loud and rasping. The child has difficulty in breathing, amounting to orthopnoea, and its face is congested. These symptoms continue for a variable period; usually they last for only one or two hours, but rarely they may continue for many hours. In one very uncommon case which was under my care the attack lasted for three or four weeks, during which time it often seemed as though suffocation was imminent. There was in this case no evidence of any lesion beyond a catarrhal laryngitis, and recovery finally took place. These attacks are partly due to obstruction in the larynx from the swollen mucous membrane, but are largely the result of a neurosis due to a highly sensitive condition of the mucous membrane. On the following day the hoarseness may continue, but the child may seem bright and may play about as usual. It is very common for the attack to recur on the second night with greater severity, but in certain cases one attack terminates the disease, and after a variable period of days, the voice becoming clearer each day and the temperature returning to normal, the child recovers. Children who have once had attacks of this kind are liable to have a recurrence until they grow older.



**DIAGNOSIS.**—The diagnosis of acute catarrhal laryngitis is to be made from foreign bodies in the larynx, traumata, and membranous laryngitis. The symptoms in the first two are not preceded by catarrhal symptoms elsewhere, which are almost always met with in catarrhal laryngitis. In a typical case of acute catarrhal laryngitis with suffocative symptoms the diagnosis is not difficult. The acute, sudden onset of the attack in the night, the loud, metallic cough, and the heightened temperature, are distinctive from the moderate temperature and the slow, progressive stenosis caused by the formation of a membrane in the larynx.

**TREATMENT.**—The treatment of acute catarrhal laryngitis is to keep the child in a room of an equable temperature of about 20° to 21° C. (68° to 70° F.) until its temperature has become normal and the hoarseness has disappeared. I have also found that a few drops of wine of ipecac, given in the latter part of the afternoon and just as the child is going to sleep, are of benefit in preventing the spasmodic, obstructive symptoms which I have just described as occurring in the night. When the attack occurs in the night the symptoms of suffocation can be best relieved by a dose of from ten to fifteen drops of wine of ipecac, or an amount sufficient to nauseate slightly. An emetic will sometimes cut short an attack of this nature, but in many cases is not necessary. An amount of ipecac sufficient to nauseate slightly, but not to cause the child to vomit, will often so relax the spasm of the larynx that the attack will soon be relieved. In many cases, however, even if vomiting has been produced, the attack continues, and other measures for relief are required. In addition to the ipecac, moderate doses of *tinctura opii camphorata* may be given. An atmosphere of steam usually gives great relief to the spasm.

Acute laryngitis is a self-limited disease, and one in which the prognosis is almost invariably good. In children who are very weak and debilitated the interference with their respiration may prove to be serious, but these cases are rare and should be treated with stimulants until the disease has run its course. The symptoms of acute catarrhal laryngitis are so terrifying to the parents that the physician is often led to look upon the disease more seriously than is necessary. Many accidents have occurred from the improper management of the steam, from giving such emetics as turpeth mineral, and from the exhibition of strong drugs, the use of which is uncalled for. The necessity for operative measures rarely arises.

**Chronic Laryngitis.**—A chronic form of laryngitis occurs in both infants and children. Syphilitic infants, as I have already told you, are at times affected by chronic laryngitis. It may also occur in tubercular disease, but is not common. Where an acute laryngitis has occurred a number of times, or where an attack has been much prolonged by improper treatment, chronic laryngitis may result. In many of these cases the voice, on the slightest exposure to dampness, becomes hoarse, and this hoarseness, after a time, may be continuous.

The treatment is to apply astringents to the pharynx, which is almost

universally involved, and to regulate the climatic surroundings of the child. Local applications to the larynx in these cases are seldom necessary.

**Pseudo-Membranous Laryngitis.**—A pseudo-membrane in the larynx may be caused by the inhalation of irritating vapors, or by the inspiration of corrosive liquids. These accidents are so readily recognized that there is no difficulty in determining the cause of the pseudo-membrane in these cases. Treatment for the relief of the stenosis should be instituted at once. This consists in the application of cold and such soothing inhalations as 3.75 c.c. (1 drachm) of compound tincture of benzoin in a quart of boiling water. The complicating œdema which is often present in these cases may require operative interference.

The most common cause of pseudo-membranous laryngitis, and the one which probably in all cases produces it, is some form of micro-organism. These micro-organisms, as I have already stated in my lecture on diphtheria, may be of several varieties. Until it is proved not to be so, however, pseudo-membranous laryngitis must be clinically looked upon as infectious and due to the Klebs-Loeffler bacillus. I must again impress upon you the fact that a simple catarrhal inflammation localized in the larynx may be produced by the Klebs-Loeffler bacillus. Pseudo-membranous laryngitis may then, until further investigations prove the contrary, be defined as an infectious inflammation of the mucous membrane of the larynx accompanied by a pseudo-membranous exudation, which may be caused by a number of micro-organisms, of which, according to our present knowledge, the Klebs-Loeffler bacillus is the most common.

I have described the symptoms, diagnosis, and treatment of pseudo-membranous laryngitis in a previous lecture (page 824), and shall, therefore, refer you to what I then said.

Some aid in the differential diagnosis of pseudo-membranous from acute catarrhal laryngitis can be obtained from the temperature, which in the latter is considerably raised, while in the former it is moderate and sometimes normal or subnormal. The slow course of a constitutional disease gradually causing obstruction is significant of this infectious form of laryngitis.

**TRACHEA.**—Pathological conditions of the trachea not connected with those of the air-passages above or below it are uncommon. The lesions of the trachea may be primary or secondary. In the latter they are merely an extension of the disease from the larynx or the bronchi, and do not play an especially significant part in the attack. The only primary disease of the trachea which is common in infancy and childhood is an acute inflammation occurring in its mucous lining. When this inflammatory condition is present, it produces an irritating cough which can usually be excited by gentle pressure over the trachea,—about the only method by which we can locate the disturbance.

The treatment is to protect the child from an atmosphere which is either too hot or too cold, from high winds, and from dust. Douching the front of the neck with cold water several times during the day is also desirable.



## LECTURE XLIX.

## DISEASES OF THE LUNGS.

BRONCHITIS.—BRONCHO-PNEUMONIA.—ATELECTASIS.—LOBAR PNEUMONIA.—GANGRENE.—TUBERCULOSIS.—PERTUSSIS.—ASTHMA.—PERIODIC CATARRH.

**LUNGS.**—The diseases which affect the lungs in infancy and childhood differ somewhat from the same diseases occurring in later life, on account of the differences which exist in the anatomical conditions at birth and during the early years of life, especially the first five. These differences I have described to you in previous lectures (pages 43 and 76). I then told you that the principal differences were that the bronchi occupied a relatively larger portion of the lung in the child than in the adult, that in the former the interstitial tissue was present in a larger amount, that the cavities of the air-vesicles were smaller, and that their walls were relatively thicker; also that the epithelial cells lining the air-vesicles were very numerous. These cells in inflammation tend to rapid cell-division, which is one of the characteristics that mark the pneumonia of childhood. These anatomical differences are of great significance when any part of the lung is diseased, and tend to make a congested lung of much more serious import in the young child than in the adult. I shall not attempt to describe to you all the various pathological conditions which may occur in the child's lungs, but shall restrict myself to those clinical groups of symptoms which represent the especial diseases. In order to do this I shall designate the disease according as the bronchi, the alveoli, or other parts of the lungs are most affected. You must remember that post-mortem examinations often show various lesions which during life were not represented by any definite symptoms, so that we cannot expect the clinical diagnosis to include entirely the pathological lesions. Beginning with the part of the lungs which is a direct continuation of the larynx and the trachea, I shall first speak of bronchitis.

**BRONCHITIS.**—Bronchitis is often secondary to some other disease, or to a direct extension from an inflammatory condition of the upper air-passages. In a number of cases, however, the group of symptoms by which we determine that bronchitis is present is so prominent from the very beginning of the attack that clinically we can describe a primary bronchitis.

By bronchitis we mean an inflammation of both the large and the small bronchi, with the exception of the ultimate divisions which lead directly into the alveoli, and which probably are never affected without involving the alveoli also. The disease may be acute or chronic.

The anatomical peculiarities of the mucous membrane lining the bron-

chial tubes—namely, the prominence of its capillaries and its comparatively loose connection to the muscular walls—render the bronchial mucous membrane peculiarly susceptible to congestion. Exposure to sudden atmospheric changes, especially humidity, appears to be of great etiological importance in the production of bronchitis. Any impurity of an irritating nature in the inspired air may in certain individuals result in an attack of bronchitis. A catarrhal inflammation of the upper air-passages is often followed by a similar inflammation of the bronchial mucous membrane. Bronchitis is of frequent occurrence in pertussis and measles. It is in children often a prominent symptom of typhoid fever, and is a frequent complication of pulmonary tuberculosis and epidemic influenza. There are also certain diseases of nutrition in which bronchitis frequently occurs. The most prominent of these is rhachitis, in which the complication of bronchitis is often of serious import.

**Acute Bronchitis.**—**PATHOLOGY.**—The pathological conditions which are present in acute catarrhal bronchitis are, according to Delafield and Prudden, a congestion and swelling of the mucous membrane, and an arrest of the functions of the mucous glands. Later, the mucous glands resume their functions with increased activity, the congestion diminishes, there is an increased desquamation of epithelium, an increased formation of the deeper epithelial cells, a moderate emigration of white blood-cells, and sometimes the red blood-cells also escape through the vessels. The whole process is a superficial one, and does not produce any change in the walls of the bronchi beneath the mucous membrane, unless it has persisted for some time, when there may be a slight thickening of the walls. When the inflammation involves the smaller bronchi they may be occluded. The occlusion of the smaller bronchi may result in the collapse of the group of air-vesicles to which they lead, and thus will be produced areas of atelectasis, which may be further changed by inflammatory processes. The bronchial glands are frequently enlarged, even in mild attacks of bronchitis.

I have here the section of a lung (Fig. 133, page 956), made by Northrup, taken from a child, which shows the exudative inflammation of the bronchi which occurs in acute bronchitis.

The specimen shows hyperplasia of the lymph-glands due to bronchitis. This condition is very commonly found in bronchitis, especially when it occurs in debilitated children. There is desquamation of the epithelium lining the bronchi, as well as a slight thickening of their walls.

**SYMPTOMS.**—The onset of acute bronchitis is usually mild, but I have seen in a debilitated infant a simple, uncomplicated bronchitis begin with a convulsion. The symptoms are very variable in their intensity, and are usually more acute and definite in a previously healthy child than in debilitated children, in whom they are often subacute and of an insidious nature. In infants and young children the bronchitis is almost always preceded by a catarrhal condition of the upper air-passages. In the mild cases there is a heightening of the temperature,  $37.7^{\circ}$  to  $38.3^{\circ}$  C. ( $100^{\circ}$  to



101° F.), cough of greater or less severity, and a slight lessening of the appetite. On physical examination the pulmonary resonance is found to be normal. A few sibilant and sonorous râles are heard with especial frequency in the area between the scapula and the vertebral column. Moist râles may also be heard. In severe cases the children suffer from more or less discomfort, produced probably by the thoracic pain, although in young children the locality of the pain cannot, as a rule, be determined. The cough is hard and dry, the respirations may be slightly raised, and the

FIG. 133.



Br., bronchus; Art., artery; Lym. Gl., lymph gland.

pulse quickened. The children may appear quite sick for two or three days, and the temperature may rise as high as 38.8° or 39.1° C. (102° or 102.5° F.); but when this latter point is reached the onset of a bronchopneumonia should be carefully watched for, especially if after from twenty-four to forty-eight hours the temperature does not fall to 37.7° or 38.3° C. (100° or 101° F.).

After a few days the severity of the symptoms lessens, the cough becomes looser, the râles gradually disappear, and under favorable conditions the symptoms subside entirely in a week or ten days. There is seldom any expectoration in children under six or seven years. In the more severe cases the râles are more numerous than in the mild form of the disease, but are of the same character. In the course of some cases of bronchitis a temporary localized diminution or even absence of the respiratory sound may result from the occlusion of a bronchus. This is especially common in infants, and ordinarily is not accompanied by a change in the percussion-sound. This form of bronchitis is the one which affects the larger and the medium-sized bronchi.

There is no characteristic temperature in bronchitis. As a rule, it is moderate,  $37.2^{\circ}$  to  $38.3^{\circ}$  C. ( $99^{\circ}$  to  $101^{\circ}$  F.), but it varies greatly according to the individual and to the degree of nervous excitement.

DIAGNOSIS.—The diagnosis of the ordinary cases of acute bronchitis, where only the large- and medium-sized bronchi are affected, is not difficult, the only disease for which it is likely to be mistaken being broncho-pneumonia. In this latter disease the greater severity of the symptoms and the higher temperature will usually show its presence, even though the physical signs may be only those which I have described as occurring in bronchitis. In the more severe forms of bronchitis it is sometimes exceedingly difficult to make the differential diagnosis from broncho-pneumonia. If, however, the temperature, after three or four days, remains high, and rises to  $39.1^{\circ}$  or  $39.4^{\circ}$  C. ( $102.5^{\circ}$  or  $103^{\circ}$  F.), with marked remissions and exacerbations, the diagnosis becomes doubtful, and in these cases we should strongly suspect that a broncho-pneumonia has arisen as a complication. We must, however, remember that in certain cases of broncho-pneumonia the temperature may be as moderate as in acute bronchitis, and we must therefore rely on a combination of symptoms rather than on any one symptom or sign. An important point in the differential diagnosis between bronchitis and broncho-pneumonia is that the physical signs in the former are much more frequently found in all parts of the thorax, while in the latter circumscribed groups of râles are often detected in different parts of the lungs. The râles in themselves, however, are not distinctive, as the râles in broncho-pneumonia are mostly those of the accompanying bronchitis. Although the physical signs of dulness and bronchial respiration are conclusive evidences that the case is not one of bronchitis alone, yet an absence of these signs does not justify us in excluding broncho-pneumonia. Where the dyspnoea, general prostration, and restlessness are slight and the temperature moderate, the case is likely to be one of bronchitis, while if these symptoms are marked, and are combined with cyanosis, dilatation of the *alæ nasi*, and a higher temperature, at least a provisional diagnosis of broncho-pneumonia should be made. In some cases the differential diagnosis will also have to be made from the onset of a pleuritis or of a lobar pneumonia, but the moderate temperature and respirations, the normal percussion-sounds, and the diffuse bilateral râles in bronchitis usually make the diagnosis from these diseases quite evident.

PROGNOSIS.—The prognosis, where no complication arises and the child is previously healthy, is good. In debilitated children, and especially where rhachitis is present, even a mild form of bronchitis may prove to be serious, on account of the danger of a complicating broncho-pneumonia, and in these cases the prognosis is much more unfavorable.

TREATMENT.—The treatment of acute bronchitis is essentially hygienic. The child should be confined to a warm, well-ventilated room which has a sunny exposure, and which is heated by an open fire to a temperature of about  $20^{\circ}$  to  $21.1^{\circ}$  C. ( $68^{\circ}$  to  $70^{\circ}$  F.). A few drops of wine of ipecac should be given if the cough is unusually dry, and to this a few drops of tinctura opii



camphorata may be added if the patient is excessively nervous. These remedies are all that will usually be needed in an attack of acute bronchitis. Where a rachitic child or one who is much debilitated is attacked by the disease, especial care must be taken to support its strength by stimulants and food.

Besides the acute bronchitis which I have just described, I have met with a class of cases which are extremely rare, but which, apparently, are instances of an exacerbation of an ordinary bronchitis through the involvement of the smaller bronchi, *not the terminal ones*. I have seen only six of these cases. These, from their clinical history, seem to have been cases of bronchitis rather than of broncho-pneumonia. I speak of them separately, as the symptoms differ somewhat from those of an ordinary bronchitis. This form of bronchitis has no connection with what was formerly erroneously called capillary bronchitis, but which is now well known to be only an early stage of broncho-pneumonia. This form of bronchitis in my cases has commonly occurred in infants in the first two years of life, though I have met with it as late as the third year. The cause, so far as could be ascertained, was the same as in an ordinary bronchitis, a catarrhal condition of the upper air-passages usually preceding the attack. The onset of the disease was rapid, and the symptoms soon became very severe. The temperature was, as a rule, moderately raised,  $37.7^{\circ}$  to  $38.3^{\circ}$  C. ( $100^{\circ}$  to  $101^{\circ}$  F.). The cough was continuous, and dyspnoea, with more or less cyanosis, rapidly developed. An examination showed normal resonance through the whole thorax, and fine moist râles. The respirations were rapid, the pulse was quick, and all the symptoms were of a violent and suffocative nature. The infants were much distressed, and were unwilling to be laid down. After from twenty-four to forty-eight hours the symptoms grew less severe, the temperature became normal or was only slightly raised, and the fine moist râles were replaced by coarse moist râles and the sibilant and sonorous râles of an ordinary bronchitis of the larger and the medium-sized bronchi.

In the early hours and days of the disease, when the symptoms are at their height, and if the infant is weak and debilitated, the prognosis is bad. If, however, the first few days are passed in safety, recovery almost invariably takes place.

This form of bronchitis is to be differentiated from broncho-pneumonia. The temperature, instead of remaining high and having the remissions of a broncho-pneumonia, soon falls so as to correspond to that of an ordinary bronchitis. The physical signs are those of bronchitis rather than of pneumonia, and the rapid recovery of the infant with the common symptoms of an ordinary bronchitis, rather than with the prolonged and characteristic symptoms of a broncho-pneumonia, verifies the diagnosis of an inflammation of the smaller bronchi.

These cases may be complicated with broncho-pneumonia, as are the ordinary cases of bronchitis.

The treatment of this class of cases is very important, as death from

exhaustion is liable to occur at any moment. The extreme congestion of the blood-vessels of the smaller bronchi may in some cases occlude the air-

## CASE 459.

## I.



## II.



Acute bronchitis. Female, 3 months old.

spaces, and areas of atelectasis may result. The indications for treatment are to oxygenate the blood, to support the strength until the disease has run



its course, and to prevent the infant from falling into a comatose condition. The treatment, therefore, is the administration of oxygen, the use of stimulants, consisting of aromatic spirit of ammonia alternating with brandy, and change of the position of the infant from time to time.

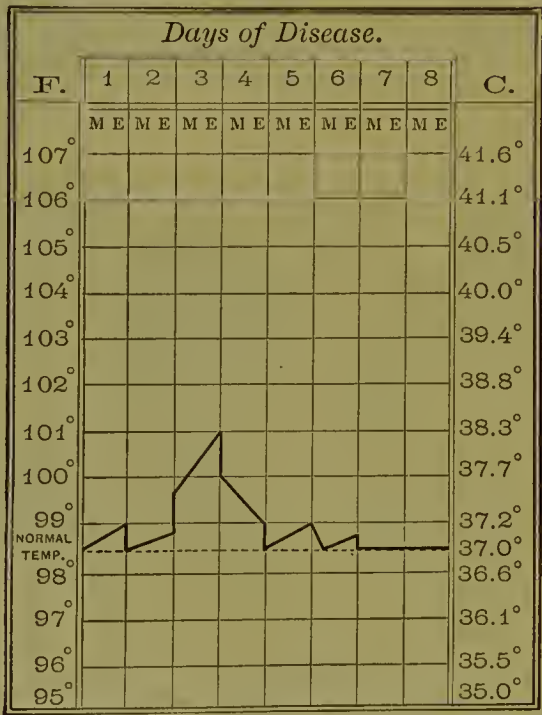
Here is an infant (Case 459, page 959), three months old, who has for the past few days had an attack of acute bronchitis, characterized by a paroxysmal, dry cough, slightly accelerated respirations and pulse, and a moderate temperature varying from 37.7° to 38.3° C. (100° to 101° F.).

The percussion of the chest has been normal, and there have been some sonorous and sibilant râles, with a few coarse moist râles heard on both sides of the chest. Early this morning the infant was attacked with excessive dyspnœa and cyanosis. Its pulse rose from 120 to 180, its respirations from 30 to 70, and its temperature from 37.7° C. to 39.1° C. (100° F. to 102.5° F.). An examination of the chest showed normal resonance and fine moist râles throughout both lungs. It has been very restless, refuses to take its food, and evidently wishes not to be laid down in its bed, but to be carried about. It is being treated with alternate doses of aromatic spirit of ammonia and brandy every half-hour. The physical signs are those of a diffuse bronchitis of the smaller bronchi, which you see I have indicated by small black spots painted on the front and back of the chest.

(Subsequent history.) After twenty-four hours the temperature fell to 38° C. (100.5° F.), the pulse to 150, and the respirations to 44. The fine râles were replaced by the ordinary coarse râles of a bronchitis, and the infant rapidly recovered.

The symptoms and course of all these cases are very similar, so that I shall speak only of one other child, whom I saw in consultation with Dr. Horace Marion, of Brighton.

CHART 32.



Acute bronchitis—exacerbation. Male, 7 months old.

A male (Case 460), seven months old, and previously healthy, for two days had a slight cough, with a few sonorous râles in the chest and a temperature varying from 36.6° to 37.2° C. (98° to 99° F.). On the third day of the attack he was suddenly seized with increased cough, dyspnœa, cyanosis, respirations of 70, a pulse of 160, and a temperature of 38.3° C. (101° F.). An examination of the chest showed normal resonance and fine moist râles

throughout both lungs. The infant was treated with aromatic spirit of ammonia and brandy in alternate doses. On the following day the temperature fell to 37° C. (98.6° F.), and the fine râles were replaced by coarse râles and sonorous râles. The bronchitis lasted for a few days, and the infant then recovered entirely.

Here is the chart (Chart 32, page 960) which shows the sudden rise of temperature.

**Chronic Bronchitis.**—Chronic bronchitis may result from a series of attacks of acute bronchitis, or from a number of other causes. Among these may be mentioned various affections of the lungs, such diseases connected with malnutrition as rhachitis, and prolonged attacks of pertussis.

The pathological conditions occurring in chronic bronchitis vary greatly in degree, and the lesions found at the post-mortem examination are often slight in comparison with the severity of the symptoms during life. In most cases there is a considerable production of mucus, pus, and serum. In cases which have lasted for a long time, in addition to the inflammatory products affecting the walls of the bronchi there may be dilatation of one or more bronchi, and the muscular coat may be thickened or thinned. Emphysema may also result.

The symptoms of chronic bronchitis are very much the same as those of acute bronchitis, except that the temperature is not so apt to be heightened, while the general symptoms of malaise, anorexia, and loss of weight are more prominent. In severe and prolonged cases where emphysema is present, the thorax may assume the position of full inspiration, the ribs being permanently raised and the antero-posterior diameter of the chest increased. The physical signs are the same as in acute bronchitis, so far as the râles are concerned. The resonance is usually normal except where the chronic process has produced emphysema, in which case there will be areas of hyper-resonance often associated with a tympanitic tone. Occasionally atelectasis of considerable areas of the lungs may take place, with a resulting lessening of the respiratory sound. This occurrence may in some cases prove to be serious, but in others the accompanying symptoms are mild, and the alveoli may again return to their normal degree of inflation.

The differential diagnosis is to be made from chronic affections of the lungs in which the thickening of the interstitial tissue has taken place with a resulting lessening of resonance, and from the condition in which the bronchi are dilated. In the latter case there are accompanying symptoms of a profuse exudation of purulent matter.

There is one form of bronchitis which from its duration may be called chronic, and yet which from the very slight degree of constitutional symptoms that accompany it corresponds rather to a subacute affection. In these cases, which usually occur in infancy and in early childhood, the child often appears quite well, but for long periods of weeks, or whenever it is exposed to a damp atmosphere, a loud wheezing will be heard in the chest. Auscultation will reveal the presence of sonorous râles everywhere, and in this variety, as well as in other forms of chronic bronchitis, a roughened sensation may sometimes be felt on palpation during respiration.



The prognosis of chronic bronchitis varies according to the cause. Where it is secondary to disease of some other organ, it depends entirely upon the prognosis of that disease. In rachitic children the prognosis is unfavorable, and in them a broncho-pneumonia is especially liable to develop, with a fatal issue. Cases of chronic bronchitis are also liable to be invaded by the bacillus tuberculosis. In cases which are the result of acute bronchitis in individuals otherwise healthy, the prognosis is favorable, provided the proper treatment can be carried out. As emphysema in chronic bronchitis is rare in children in comparison with adults, the chances for recovery in the former are correspondingly good.

The treatment of chronic bronchitis is essentially climatic. The children should be kept in a warm dry climate for some months after the bronchitis has entirely disappeared. Especial care should be taken that the child is suitably protected by flannel undergarments. Where other treatment is required, as a rule, tonics will prove of more benefit than the drugs which are usually administered for their direct effect upon the bronchial mucous membrane.

**FIBRINOUS BRONCHITIS.**—During the course of what may appear to be an ordinary bronchitis, in rare instances a fibrinous form of bronchitis has been met with. In this variety masses of fibrin in the bronchi form casts of various extent according to the number of the bronchi which are affected.

The disease may run a short course of days or weeks, but is usually chronic and may last for years. The paroxysms may also be periodic.

The diagnosis can be made only when portions of the casts have been expectorated.

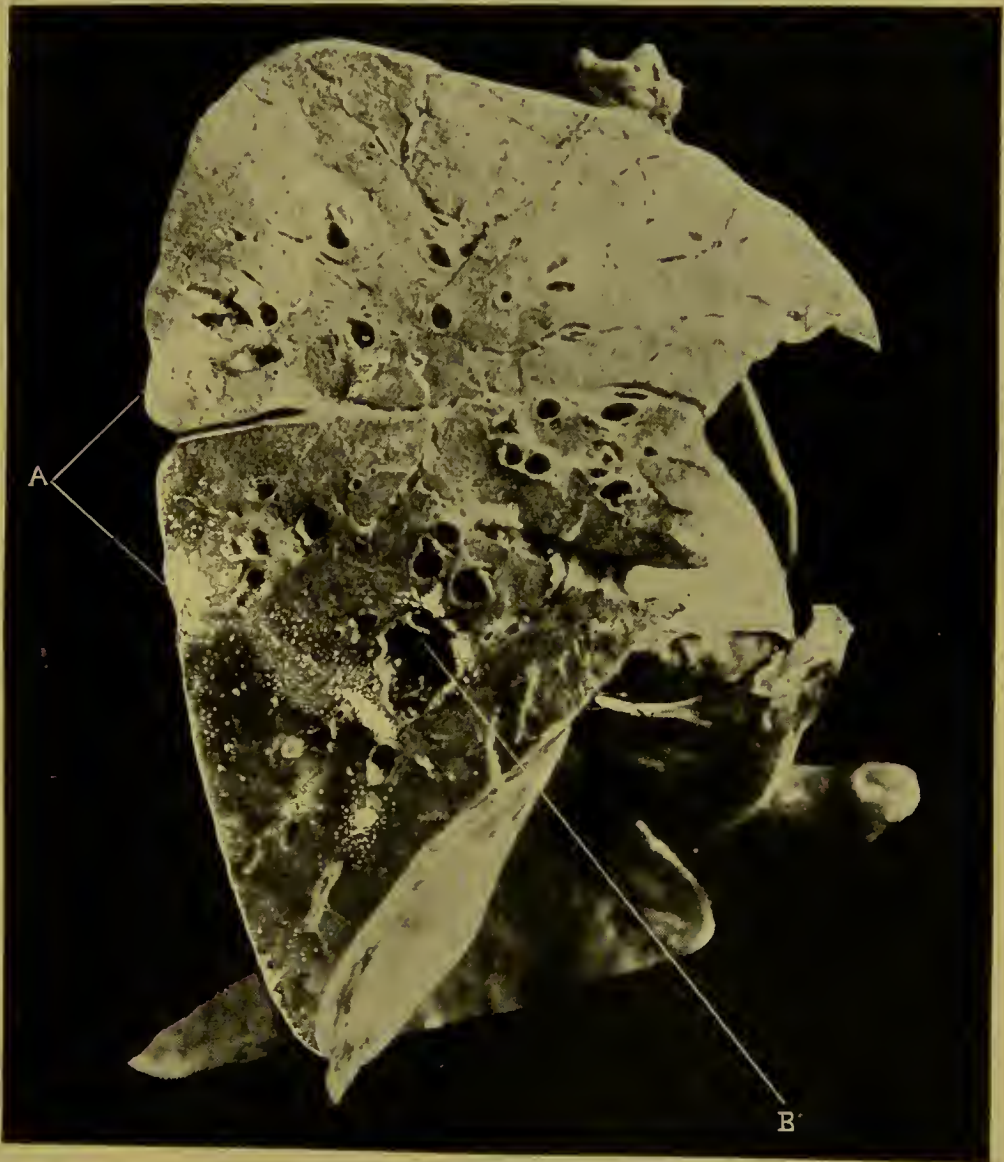
The treatment is chiefly by the inhalation of steam or of atomized lime water, and by supporting the strength with proper nourishment and stimulants until the disease has run its course.

**BRONCHO-PNEUMONIA.**—Broncho-pneumonia is an affection of the lung characterized by an inflammation of the walls of the terminal bronchi and of the alveoli. The disease may be acute or chronic. It may occur at any age, but is the most common and fatal form of inflammation of the lung during the first five years of life, and is much more fatal than lobar pneumonia at this period. During this early period, and especially during the first two or three years of life, the lung, from its embryonic type, is more frequently subject to the form of inflammation occurring in broncho-pneumonia than at a later and more developed period. The disease is usually secondary to bronchitis, and commonly occurs in connection with measles, scarlet fever, pertussis, and diphtheria. Broncho-pneumonia is also a very important disease, not only as grave in itself, but also because it is so frequently followed by tuberculosis.

**ETIOLOGY.**—A prominent predisposing cause of broncho-pneumonia is age, and where pneumonia occurs in a child under five years of age it is usually in the form of broncho-pneumonia. This is due principally, as I have already stated, to the anatomical conditions met with in early life.

Children who are weak or debilitated by previous diseases show a predisposition to broncho-pneumonia, and it therefore frequently arises in the course of tuberculosis, chronic gastro-enteric diseases, and rhachitis. Those seasons of the year which are marked by cold, moisture, and variations of temperature especially predispose to the development of broncho-pneumonia. All these conditions, however, in all probability merely prepare the way for the entrance of certain germs which produce the disease. What these micro-organisms are is still uncertain, as it is known that a number of different organisms can produce the disease. The origin of broncho-pneumonia from intestinal infection must also be considered. (Sevestre.)

FIG. 134.



Acute broncho-pneumonia involving different areas of the lung. A, consolidated lung-tissue ; B, dilated bronchus. (Warren Museum, Harvard University )

**PATHOLOGY.**—In broncho-pneumonia the inflammatory process affects the walls of the smaller and terminal bronchi, which become thickened and markedly infiltrated with cells. The inflammatory process then extends through the walls of the bronchi to the surrounding air-vesicles as well as to the terminal ones. In this way centres of consolidation are formed in



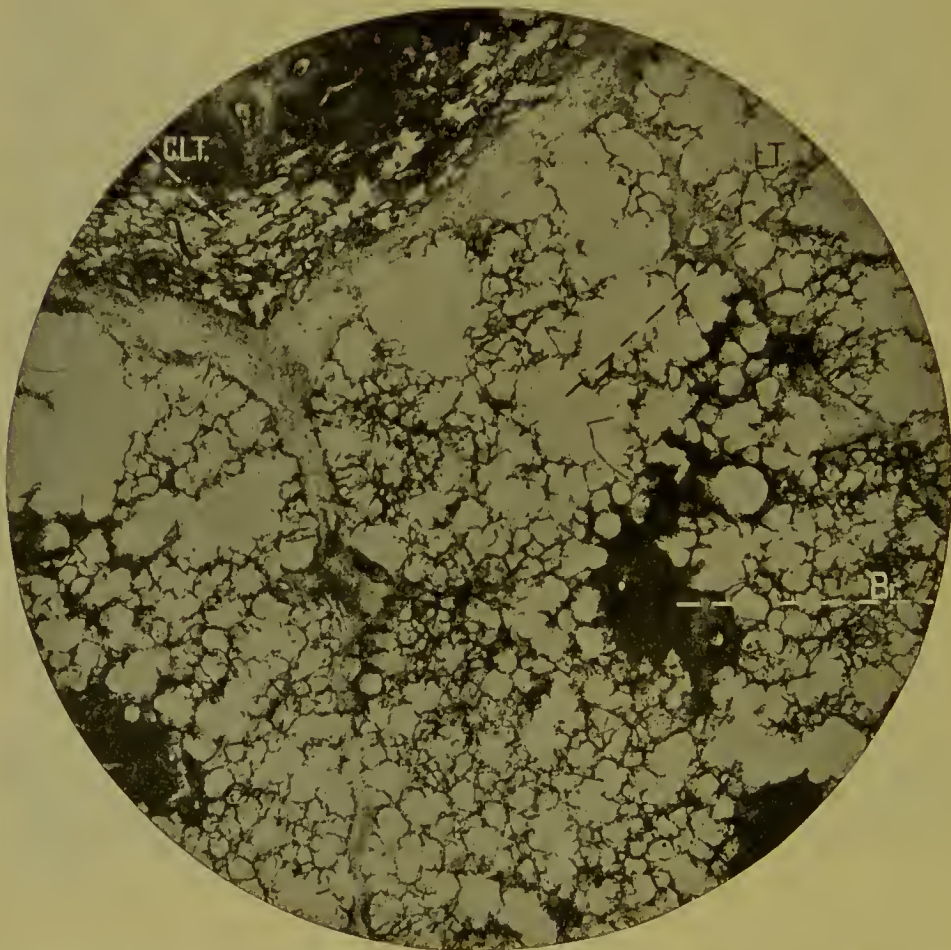
different parts of the lung. The course of this inflammation varies in its rapidity, at times attacking only a small portion of the lung, and again being more diffuse in its onset and gradually invading large areas. The lesions are irregular in their distribution, and usually occur in both lungs. They are at times so extensive as to involve a whole lobe, but, as has been stated by Northrup, whatever the extent of hepatization, whatever the time occupied in its course, and whatever the post-mortem appearances, the essential lesion is an inflammation of the walls of the terminal bronchi and of the adjacent alveoli.

This lung (Fig. 134, page 963), taken from a young child, presents the macroscopic lesions of broncho-pneumonia.

You will notice that the areas of consolidation surround the bronchi, and that this bronchus (B) is markedly dilated.

This section of a lung (Fig. 135), made by Northrup, was taken from an infant sixteen months old, in whom the broncho-pneumonia was a complication of measles. It shows the early pathological lesions of broncho-pneumonia.

FIG. 135.



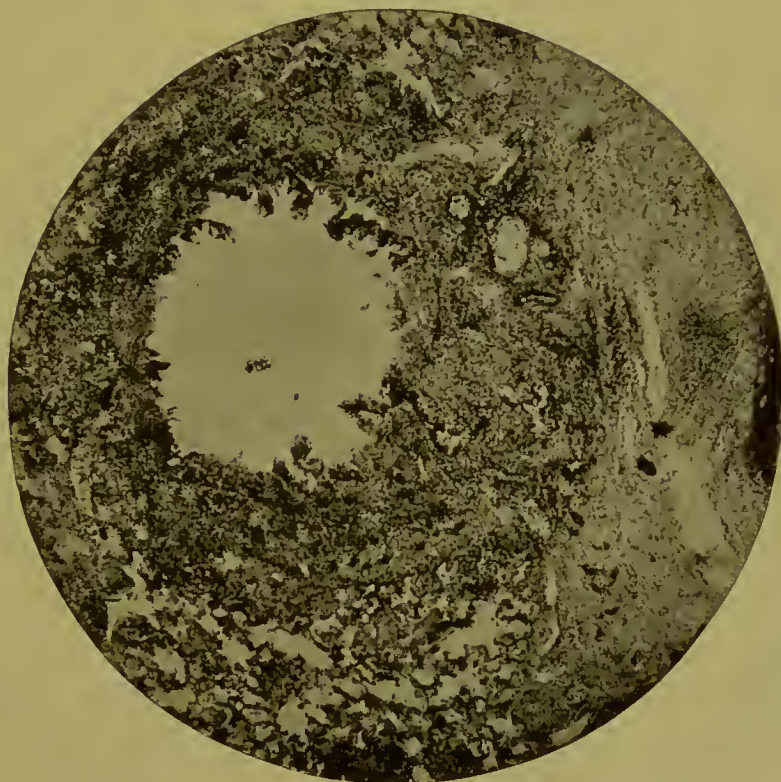
Broncho-pneumonia complicating measles. Early stage. C. L. T., consolidated lung tissue; Br., bronchiole; L. T., emphysematous lung tissue.

You will notice that in one of the lobules there are two bronchioles (Br.) with infiltrated walls and pus within them. They are also filled with exudation, and the lumen of each is almost entirely occluded. A portion

of a neighboring lobule is consolidated (C. L. T.). A considerable portion of the lung tissue (L. T.) in the section is, as you see, normal or emphysematous.

Here is another section (Fig. 136), made by Northrup, illustrating the broncho-pneumonia which follows a diphtheria descending from the upper air-passages, and which may occur in any acute infectious disease. It shows the typical lesion of broncho-pneumonia.

FIG. 136.



Broncho-pneumonia secondary to diphtheria.

In the bronchus, which you see enlarges in the middle of the section, the lining mucous membrane is hanging in shreds into its lumen. The walls of the bronchus are densely infiltrated, and the contiguous alveoli are filled with exudation to a greater or less extent and are consolidated by it. This section was taken from the lung of a child three years old who during an attack of scarlet fever developed diphtheria of the larynx. The diphtheritic process descended later into the bronchi.

Here is another section (Fig. 137, page 966) taken from the lung of the same child, but showing the tissue relatively less affected.

Many of the consolidated alveoli contain free blood-cells. The bronchial wall (Br.) is infiltrated and almost entirely denuded of its lining membrane.

These smaller bronchi are surrounded by zones of intense congestion and infiltration. When the inflammation is intense and is accompanied by abundant secretion these bronchi frequently become dilated. This dilatation is associated with a weakened condition of the bronchial walls and with an abundant secretion. These dilatations probably, according to the



observations of Northrup, wholly disappear on the recovery of the patient. As has already been described in the pathology of bronchitis, the bronchial lymph-glands are always enlarged in broncho-pneumonia, and there may be fibrin on the pulmonary pleura. According to Delafield, in the zones of peribronchitic pneumonia the walls of the air-vesicles are thickened or swollen, either with or without some cellular infiltration, and the cavities of the air-vesicles are filled with epithelial cells and pus-cells, with fibrin and

FIG. 137.



Broncho-pneumonia secondary to diphtheria. Br., bronchus; C. L. T., consolidated lung-tissue; N. L. T., lung-tissue nearly normal; Art., artery.

red blood-corpuscles in varying proportion and amount. Fibrin when present is only in small quantities, and often is absent altogether. The capillaries in the walls of the vesicles are congested and prominent. The portions of lung which are not hepatized are congested and cedematous. The cavities of the air-vesicles are diminished by the enlarged capillaries and the swollen vesicular epithelium.

In addition to the other lesions which I have just described, areas of atelectasis are frequently found in broncho-pneumonia. This atelectasis is usually produced by mechanical causes, such as obstruction by pus or tenacious mucus. It may also arise as a result of enfeebled respiratory power. The blood-vessels become dilated, the walls of the alveoli partially collapse, the residual air is absorbed, and an exudation of serum with proliferative

cells and leucocytes takes its place. The atelectasis is commonly symmetrical, affecting the posterior margin of both lower lobes of the lung, but it may also appear in irregular scattered areas in the posterior portions of the upper lobes (Northrup). It may occur either during the acute stage of the inflammation or later when the pneumonia has become chronic. There are no distinct stages in the pathology of broncho-pneumonia which correspond to those of lobar pneumonia. According to Northrup, broncho-pneumonia develops by the irregular invasion of successive portions of the lungs, and the process resolves in like manner. The different consolidated areas in the same lung may often show all the stages. The mottled appearance which is so often noticed macroscopically in these lungs may be caused by the presence of lobules of gray and red hepatization lying side by side. Of these inflammatory products the fibrin disintegrates quickly, and is therefore absorbed more rapidly than the cellular elements, which do not disintegrate so readily. In lobar pneumonia, therefore, absorption takes place sooner than it does in broncho-pneumonia, where the products of inflammation are mostly cellular and resolution and absorption are naturally slow.

Instead of the gradual disappearance of the various pathological lesions the pneumonia may persist. This persistent form of the disease may, according to Delafield and Prudden, follow a single attack of acute broncho-pneumonia, or there may be several acute attacks before the chronic condition becomes evident, and the course of the disease may thus vary in different cases. When this persistent broncho-pneumonia occurs, the proliferative cells take part in the formation of new connective tissue, and in this way persistent thickening is caused. The alveolar walls of certain portions may become similarly thickened. The walls of the bronchi and their surrounding tissue are especially subject to a persistent thickening and a formation of new connective tissue constituting chronic broncho-pneumonia and peribronchitis. The bronchi already dilated become still more enlarged by the contraction of the cicatricial tissue surrounding them. The uneven contraction of this new tissue, together with the pressure within the tubes facilitated by a weakened condition of the walls, allows of saccular as well as of fusiform dilatation of the bronchi. The epithelial cells of the dilated bronchi proliferate, and, falling from the bronchial walls, mix with the bronchial secretion. The remaining epithelium is swollen and loose. The lesions of chronic broncho-pneumonia are frequently associated with tuberculosis of the bronchial glands and with other tubercular lesions.

In connection with the pathological lesions occurring in chronic broncho-pneumonia a condition called *fibroid phthisis* has in very rare cases been noticed in children. The lesions which represent fibroid phthisis are manifested in the presence of connective tissue in the lung, with a corresponding destruction of the true parenchyma. These changes are usually unilateral, and should not be considered as representing a disease, since they merely occur in the course of various chronic pulmonary affections, among which are tuberculosis and chronic broncho-pneumonia.



Under this microscope (Fig. 138) you will see a section of the lung, made by Dr. Northrup, taken from a young child with chronic broncho-pneumonia.

FIG. 138.



Chronic broncho-pneumonia. N. L. T., normal lung-tissue; C. L. T., consolidated lung-tissue; Br., bronchi, some of them dilated.

You will notice the areas of consolidated lung (C. L. T., peribronchitis) around the bronchi, which are dilated (bronchiectasis). You will also observe that there are areas of normal lung-tissue (N. L. T.).

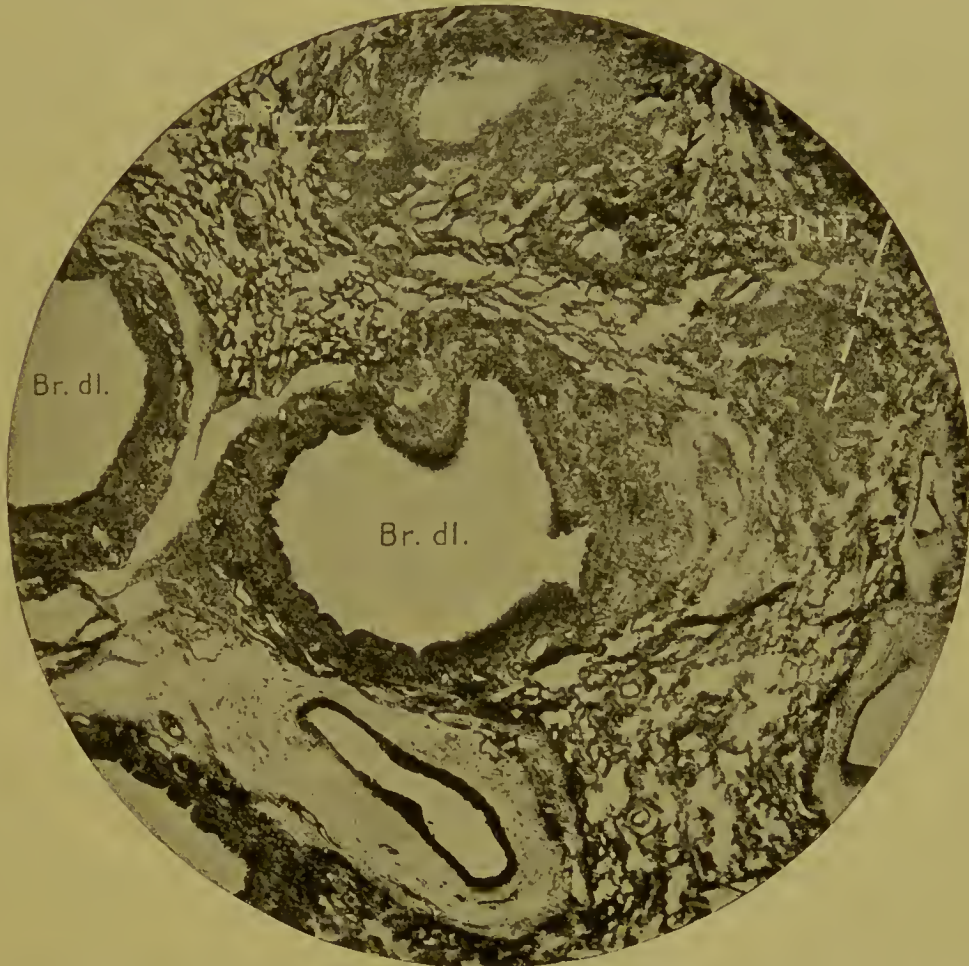
Under this second microscope (Fig. 139) is a section, also made by Dr. Northrup, taken from a lung with chronic broncho-pneumonia in which the process has advanced still further than in the other.

In the middle of the specimen you will see a dilated bronchus with a section of a blood-vessel just below it. There is considerable connective-tissue formation about both. Here you see that the process of a peribronchitic pneumonia has gone further than in the other specimen (Fig. 138), and that there is, in addition to the dilated bronchi with the surrounding cellular infiltration, a tendency to the formation of connective tissue in the interlobular septa. This is the form of chronic broncho-pneumonia which is sometimes called interstitial pneumonia, and is usually characterized by a long course and delayed recovery.

A frequent lesion which occurs in the course of broncho-pneumonia is *emphysema*. According to Northrup, it is usually vesicular and situated in

the anterior portion of the upper lobes. It is due to the diminished amount of air-capacity, together with the violent introduction of air into the chest caused by dyspnœa and coughing. This distention of the air-vesicles is supposed usually to disappear with the subsidence of the lesion which is causing the emphysema. Emphysema, both of the vesicular and of the interstitial variety, most commonly occurs in the pneumonia which follows pertussis.

FIG. 139.



Chronic broncho-pneumonia. Br. dl., dilated bronchus; Th. L. T., thickened lung-tissue; Br. Pn., broncho-pneumonia.

The interstitial variety may exist in the form of superficial sacs formed by the rupture of air-vesicles beneath lifting the pleura, or it may extend between the lobules in V-shaped tracts from the anterior edge of the upper lobe even to the root of the lung.

**SYMPTOMS.**—The symptoms of broncho-pneumonia vary greatly, owing to the many different lesions which commonly occur in the disease and which by their greater or less severity make its course exceedingly irregular. In so many instances is the broncho-pneumonia secondary to some other disease that the symptoms are necessarily modified by those of the initial affection. Thus, where broncho-pneumonia arises in the course of diphtheria, the symptoms are often obscured by the severity of the general symptoms of the diphtheria. Where broncho-pneumonia is secondary to measles and to pertussis, although at times its onset is difficult to detect, yet, as a rule, the



quick respirations, the marked and continuous rise of temperature, and the evident exacerbation in the severity of the pulmonary symptoms, usually permit a diagnosis to be made even before the physical signs have become prominent. Its onset, however, in measles is, as a rule, rapid, while in pertussis it is slow and insidious.

The group of symptoms which characterizes a broncho-pneumonia arising during the course of bronchitis is somewhat more definite. In place of the moderate temperature and the absence of signs of serious disease which are usually met with in the course of an ordinary bronchitis, when broncho-pneumonia supervenes the temperature rises, the pulse and respirations are quickened, the *alæ nasi* dilate, there is more or less cyanosis, the cough becomes more frequent and painful, and the general aspect of the patient is that of one suffering from an affection of a severe type.

The temperature in broncho-pneumonia varies greatly, according to the extent and severity of the lesions. Corresponding to the intensity of the pneumonic onset, or to the especial disease which it complicates, the temperature rises rapidly or slowly and insidiously. The most common course in mild cases with gradual onset and terminating in recovery is for the temperature to rise gradually to  $39.4^{\circ}$  or  $40^{\circ}$  C. ( $103^{\circ}$  or  $104^{\circ}$  F.), then to have a morning remission of three or four degrees for a number of days, and then to fall irregularly by lysis. A crisis is very rare in broncho-pneumonia, but sometimes occurs. Although the remissions in the temperature during the active stage of the disease are often quite marked, yet, as a rule, the temperature does not at this time fall to the normal. This is of service in differentiating certain cases of broncho-pneumonia, as well as lobar pneumonia, from malaria. Occasionally the temperature is reversed, the highest point being reached in the morning. This is rare, and is of no especial significance. Where the temperature instead of remitting remains high and steadily rises, the disease, as a rule, soon terminates fatally. Instead of the continued high temperature which occurs so often in fatal cases, a low temperature of only a few degrees above normal is sometimes met with, usually where the vitality is low and the power of reaction slight. The duration of the heightened temperature is very variable in broncho-pneumonia, and may last for a number of days or for weeks without the necessary result of the grave lesions of a more chronic process.

The pulse and respiration, though quickened, vary according to the severity of the disease and also according to the degree of nervous excitement. This latter is a very important element to be considered in determining the gravity of their rate. The pulse is at times very rapid, 160–180, and even higher; it usually varies from 130 to 150 or 160; though regular and full at first, it becomes weak and sometimes irregular as the disease progresses, and is very apt to remain rapid even after the temperature has declined and convalescence has been established. The respirations may be quickened by an unusually high temperature, but depend mostly on the extent of the involvement of the alveoli. They vary from 50 to 80, but

they may be even higher, and are accompanied by dilatation of the *alæ nasi*. The respiration often shows a pause after inspiration instead of after expiration, as occurs in normal respiration, and is usually accompanied by an expiratory moan.

This sign, however, is not characteristic of broncho-pneumonia, as it may occur in lobar pneumonia and in various affections where the circulation is interfered with and where respiration is painful. In like manner the dilatation of the *alæ nasi* may occur in any disease accompanied by a heightened temperature and nervous excitement. Temporary exacerbations and changes in the rhythm of respiration are quite common in broncho-pneumonia, and in some cases a Cheyne-Stokes type of respiration has been noticed. This sign is usually one of grave import. Recession of the epigastrium and of the intercostal spaces commonly occurs in broncho-pneumonia, and varies according to the severity of the pulmonary lesions. In infants painful respiration is shown by a frown rather than by crying, while in young children it is shown by their whimpering and suppressed cries.

The physical signs of broncho-pneumonia are almost entirely those of the accompanying bronchitis, but in typical cases they correspond to the various pathological lesions which I have just described. According as larger or smaller areas of the lung are involved, corresponding areas of dulness on percussion may be found, provided these areas are sufficiently extensive not to be masked by other resonant portions of the lungs. They can, as a rule, be detected best by very light percussion. These areas of dulness are usually bilateral and of somewhat varied extent, though, as I have already stated, an entire lobe may in rare instances be sufficiently involved by the broncho-pneumonic process to produce very extensive areas of dulness. Over the area of dulness bronchial respiration, and in some cases increased vocal resonance and fremitus, may be found. On auscultation moist râles of all sizes may be heard all over the lungs, or, as is more usual, in circumscribed areas.

A symptom which occurs quite commonly in broncho-pneumonia is cyanosis. This may not only arise from the interference with the oxygenation of the blood from the lesions involving the air-vesicles, but may also be produced by a temporary atelectasis of certain portions of the lungs. The cyanosis is often accompanied by attacks of dyspnœa. When these symptoms result from atelectasis, the temperature, as a rule, does not rise, but may even be somewhat reduced, and areas of dulness may be detected on percussion. During these paroxysms the skin is often cold and moist. When the cause of the atelectasis, whether it be obstruction by plugs of mucus or pus or temporary exhaustion of the contractile powers of certain portions of the lungs, has been removed, the cyanosis and dyspnœa pass away and the general symptoms improve. These symptoms may arise at various periods during the course of broncho-pneumonia, and unless the atelectasis passes off within a few days a fatal issue is very apt to result.

Well-marked physical signs, especially dulness on percussion, are usually



found at the bases of both lungs behind, and also between the scapulae and the vertebral column. The earliest changes, however, in percussion and auscultation are often first detected in the highest part of the axilla. These signs of consolidation are rarely found in the early days of the disease, when the bronchitic signs are usually all that can be detected. The physical signs are markedly modified when atelectasis or emphysema is present.

In cases which recover, resolution takes place slowly and the lung gradually returns to the normal condition. Great weakness and prostration often last for a long time. Relapses are quite common.

COMPLICATIONS.—Pleurisy of a light grade is not an uncommon complication of broncho-pneumonia. Abscess and gangrene sometimes, though very rarely, arise. A case of the latter occurred at the Boston Children's Hospital in the service of Dr. Morrill.

A very frequent and important complication of broncho-pneumonia is tuberculosis.

In certain cases of the fulminant type of broncho-pneumonia the post-mortem examinations show extensive deposits of miliary tubercle, which in these cases is the cause of the accompanying acute inflammation. This condition is called tubercular broncho-pneumonia.

A frequent, short, hacking, and painful cough is a constant symptom from the beginning of the disease, and even after resolution has taken place this may continue for a long period. Infants and young children, even up to the age of seven or eight years, have often not learned to expectorate, so that we cannot, as in adults, judge of the character of the sputum. When the sputum is seen it corresponds to the pathological exudation which I have just described when speaking of the pathology of the disease. Vomiting is at times met with, and diarrhoea is not uncommon. In certain cases disturbance of the gastro-enteric tract is present from the very beginning, and the intestinal disease is apparently as important a feature of the attack as the pulmonary part. As the attack progresses the child loses much in weight, the face often looks pinched, and at times during the height of the disease there is a certain amount of delirium, which in combination with other grave symptoms, such as uncontrollable diarrhoea and a depressed temperature, is a serious symptom.

DIAGNOSIS.—The diagnosis of broncho-pneumonia should first be made from the bronchitis which ordinarily accompanies it. This has already been sufficiently referred to in speaking of the diagnosis of bronchitis.

The differential diagnosis between the non-tubercular and the tubercular forms of broncho-pneumonia is important, but can rarely be made in the early stages of the disease, as the lesions are the same, and a bacteriological examination of the sputum in these cases can seldom be obtained.

The disease which should be especially considered in making the diagnosis of broncho-pneumonia is lobar pneumonia. The two diseases are perfectly distinct, in onset, course, duration, and termination, and can best be described when I speak of the diagnosis of lobar pneumonia (page 985).

PROGNOSIS.—Age is a very important factor in the prognosis of broncho-pneumonia. As Morrill has shown by a carefully prepared table, a large majority of the fatal cases of broncho-pneumonia occurs in the first two years of life. The prognosis varies according to the disease in the course of which it occurs. It is most grave when it occurs in pertussis, especially in infants, and the younger the child the more fatal the disease. Next to pertussis, the gravity of the prognosis is greatest in measles and diphtheria. When it occurs in such diseases as rhaehitis and tuberculosis, or where the individual has not been well cared for, the prognosis is also very unfavorable. I have already referred to the temperature as a prognostic sign in broncho-pneumonia. According to Holt's observations, the highest mortality occurs among the cases of shortest duration, and the disease is universally fatal when its duration is shorter than four days. After this early period of danger is passed the prognosis becomes much more favorable, the lowest death-rate in Holt's cases being met with in cases terminating in from eight to fourteen days. When the disease lasts for more than two weeks the chances of recovery are lessened every day that the temperature remains raised. The cases in which there is a very high temperature,  $41.1^{\circ}\text{C}$ . ( $106^{\circ}\text{F}$ .), are usually fatal. Where the disease is protracted, death generally occurs from exhaustion.

TREATMENT.—The treatment of broncho-pneumonia is that of the special disease to which it is secondary. The strength should be carefully supported from the time that the disease is first detected until convalescence has been completely established. The patient should be carefully nursed, as the nursing is the most important part of the treatment of broncho-pneumonia and requires much judgment and intelligence. The atmosphere of the room should be equable, the temperature from  $20^{\circ}$  to  $21.1^{\circ}\text{C}$ . ( $68^{\circ}$  to  $70^{\circ}\text{F}$ .), and especial attention should be paid to the ventilation. The heat and ventilation obtained from an open wood fire are especially valuable. As few drugs as possible should be given, since there is no drug which will cut short the disease, and most of the drugs commonly used in the treatment of pulmonary affections are, as a rule, of more harm than benefit in broncho-pneumonia. The vitality of infants and young children is so easily lessened by a disease so severe as broncho-pneumonia that the respiratory power is likewise quickly diminished, and we should avoid, except where they are especially needed, such drugs as opium. Ipecac in minute doses seems to facilitate the removal of the mucus. During severe paroxysms an atmosphere of steam or the administration of oxygen is indicated, according as the symptoms seem to be produced by a tenacious exudate or by unaerated lung-tissue. In cases where cyanosis and dyspnoea are urgent, if these depend upon mechanical obstruction with its resulting atelectasis, an emetic is occasionally demanded. In some cases, also, where much exhaustion arises from incessant coughing, small doses of tinctura opii camphorata may be used with caution, and discontinued as soon as possible. When the urgent symptoms are caused by the heightened temperature, much relief



can be obtained by reducing the temperature by means of the warm bath given at a temperature of  $32.2^{\circ}$  C. ( $90^{\circ}$  F.). This may be followed by the warm wet pack, which can often be continued with benefit for several hours, and is especially beneficial in producing deep inspirations and thus aerating dependent portions of the lung. The position of the child should be changed from time to time, as there is a tendency for the inflammatory exudate to collect in the lower and back portions of the lungs. The administration of food at regular intervals is very important, and should be carried out rigorously. In most cases the chief part of the diet, if possible, should be milk. Although vomiting may occur in certain cases, as a rule, if the diet is carefully regulated and the milk given once in two hours with stimulants adapted to the condition of the especial case, an over-sensitive condition of the stomach is seldom a serious obstacle to the treatment. In a number of cases the paroxysmal attacks of cyanosis and dyspnoea may be caused by a weak heart. In these cases the administration of brandy and digitalis, the latter in the form either of tincture or of infusion, for a few days, until the cardiac condition has improved, is indicated. Strychnine and nitroglycerin may also be used, and the former is considered especially important.

When convalescence has been established the children are often left in a very weak condition, and careful attention should then be paid to the nursing and to the general hygiene. The strength should be restored by means of tonics, and, if possible, the child should be removed to an equable, warm climate.

CASE 461.



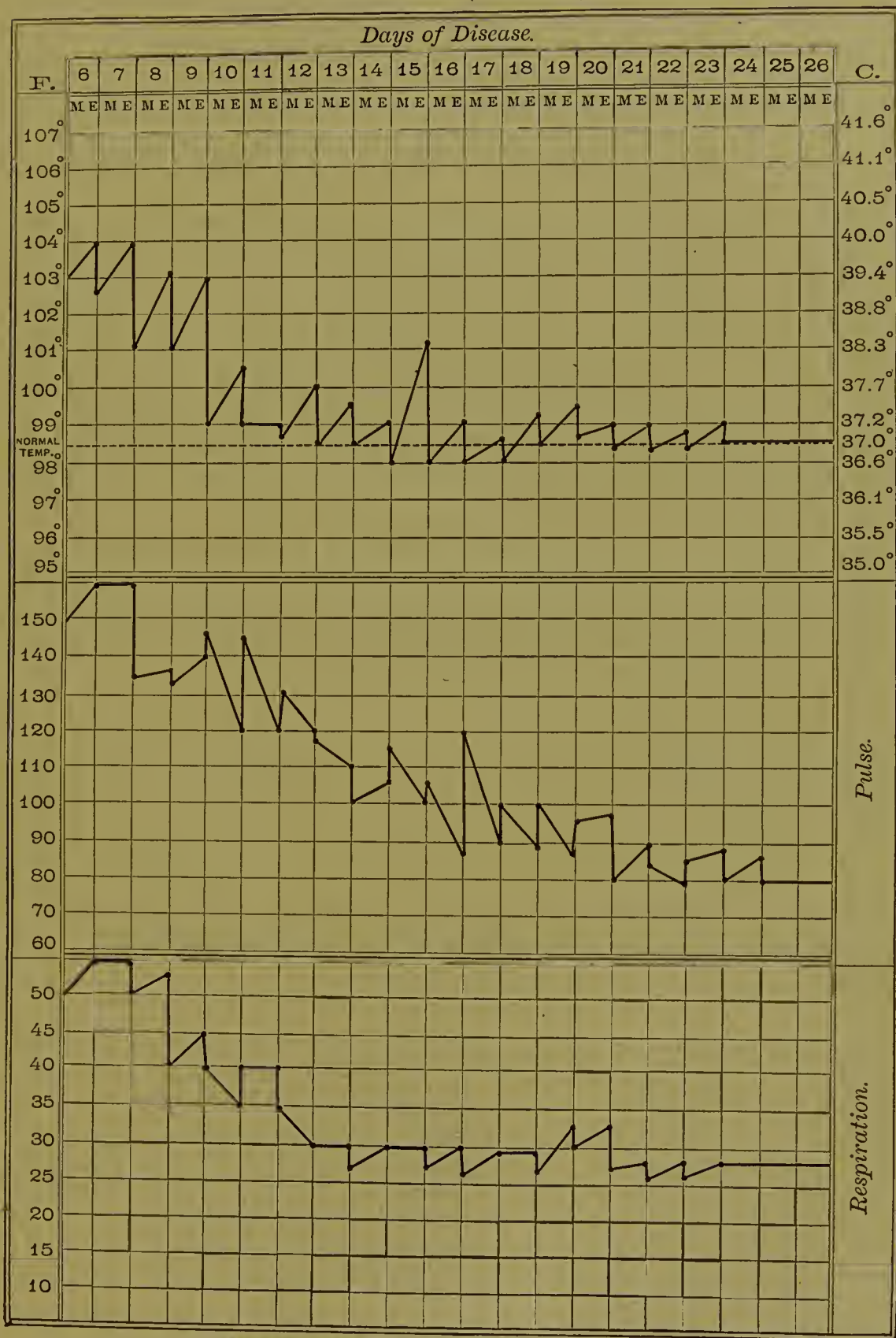
Acute broncho-pneumonia. Female,  $4\frac{1}{2}$  years old. The black circles indicate areas of consolidated lung-tissue; the black spots indicate râles.

Here is a little girl (Case 461), four and a half years old, in whom the physical examination shows very marked lesions of broncho-pneumonia.

There is no tubercular history in her family. She had scarlet fever when she was one year old, measles when she was one and a half, pertussis and varicella when she was three, and bronchitis when she was three and a half years old. She had otherwise always been

well up to nine days ago, when she began to complain of headache and pain in her chest. At that time she vomited, and two days later began to cough and to be rather somnolent.

CHART 33. (CASE 461.)



Acute broncho-pneumonia. Recovery in thirty days.

Her bowels were regular. On physical examination the child is found to be rachitic, as shown by a rosary, enlarged epiphyses of the wrists and ankles, and marked bowing of the legs. On entering the hospital her pulse was 160, her respirations 60, and her temperature



39.4° C. (103° F.) in the morning and 40° C. (104° F.) in the evening. She seemed very sick, had considerable cough, but no expectoration; there was some dyspnoea, and at times she was somewhat cyanotic. On examining the chest the percussion was found to be resonant, but throughout both lungs there were moist râles. Nothing abnormal was detected on examining the heart and abdomen. She was treated with milk and brandy.

On the following day she was in about the same condition, and her pulse, respirations, and temperature were as on entering the hospital. In certain circumscribed areas in both backs slight dulness was detected on percussion, with moist râles around the edges of these areas.

On the third day the pulse had fallen to 136, the respirations to 40, and the temperature to 38.3° C. (101° F.).

To-day, the ninth day of the disease, the pulse is stronger and the child's condition is very much improved. The dyspnoea has disappeared almost entirely, there is no cyanosis, and she is more comfortable. On examining the front of the chest you notice that the resonance on percussion is normal, and I find no abnormal sounds on auscultation. On examining the back you will find certain circumscribed areas of dulness, the borders of which I have marked in black. One of these areas is between the edge of the scapula and the vertebral column, another is at the right base in the posterior axillary region, and another is at the left base just below the angle of the scapula. Over these areas of dulness bronchial respiration is heard. Just outside of the areas of dulness can be heard in limited areas moist râles of various sizes, which I have indicated by black dots. You will notice that the physical signs in this case of broncho-pneumonia correspond to the areas where the lesions of this disease are usually detected on physical examination.

(Subsequent history.) Five days later the child was found to have much improved. During the following ten days the abnormal signs in the chest disappeared, but the pulse, respirations, and temperature did not become permanently normal for a week later. The child, after remaining weak and debilitated for some weeks, was finally discharged from the hospital in good condition. The chart of this case is seen on page 975.

This case apparently arose in the course of a slight bronchitis occurring in a rachitic child. I have told you that the prognosis of broncho-pneumonia in rachitis is usually unfavorable, but in this case the child possessed sufficient vitality not to succumb to the disease.

In connection with this case, and with what I have just said of the gravity of the prognosis of broncho-pneumonia when occurring in connection with rachitis, I shall recall to your minds the case which I lately showed you in the wards of the City Hospital.

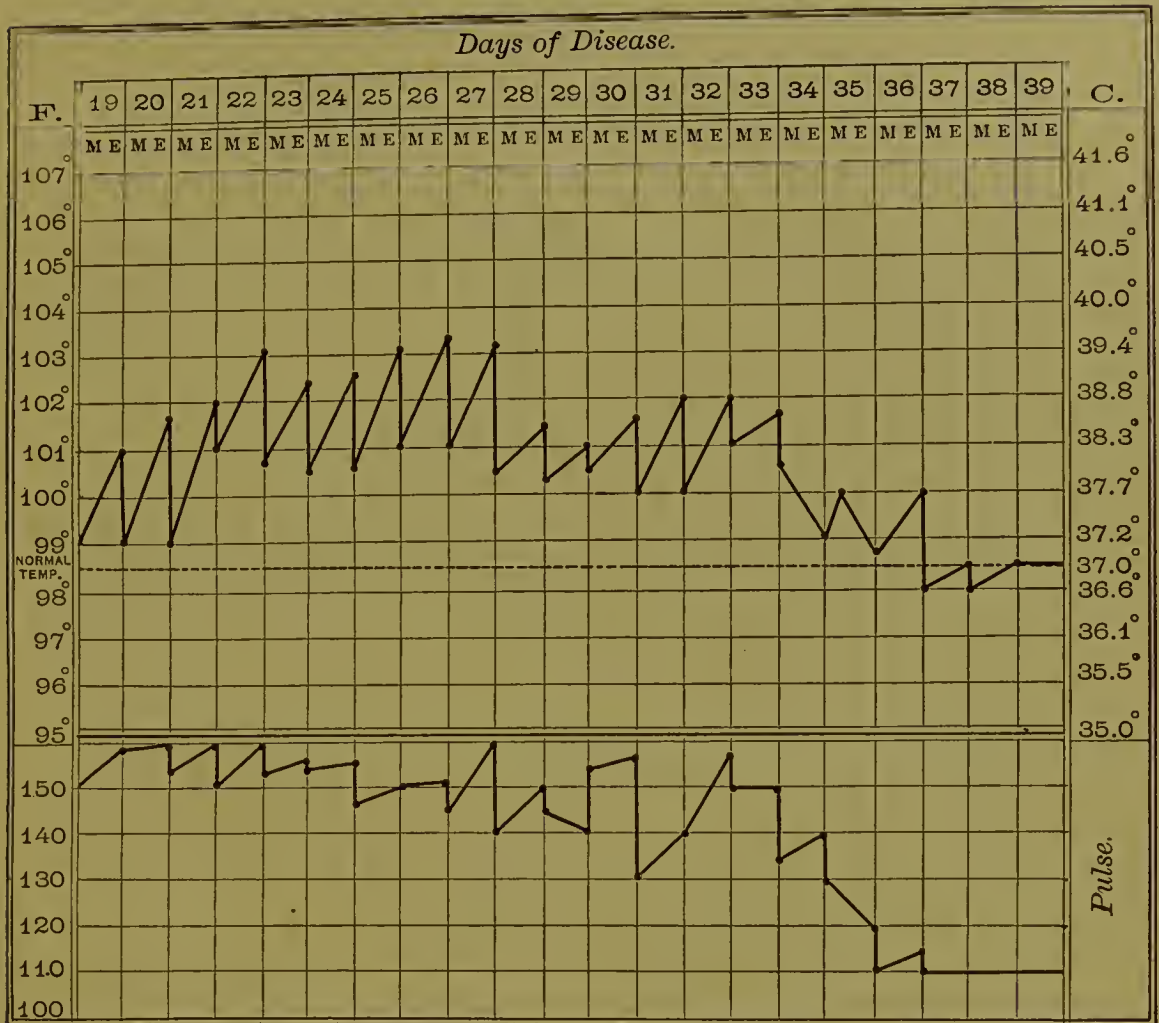
The child (Case 462) was two years and one month old. Its mother died of pulmonary tuberculosis. It had bronchitis when it was one year old, and the cough continued for three months. One week before entering the hospital it was attacked with a severe cough, and began to lose in weight and to have diarrhoea. A physical examination showed that it was a case of marked rachitis. The breathing was rapid and labored, there was considerable cyanosis, and the child was dull and somnolent. Patches of dulness were found in various parts of the lungs, with moist râles of different sizes. The pulse varied from 140 to 150, the respirations from 80 to 90, and the temperature from 38.8° to 40° C. (102° to 104° F.). The symptoms increased in severity, the child grew weaker, and on the second day after it entered the hospital it died suddenly.

When broncho-pneumonia attacks a child with such marked rachitis as was shown in this case, a fatal issue almost always results.

Here is a chart (Chart 34, page 977) showing the temperature and pulse

of an infant (Case 463) eight months old, from the nineteenth day of an attack of broncho-pneumonia until convalescence was established.

CHART 34.



Acute broncho-pneumonia. Infant, 8 months old. Recovery in thirty-six days.

The infant, a male, had always been strong and well. The parents were healthy, but on the father's side a number of brothers and sisters had died of pulmonary tuberculosis. It was being nursed by its mother, who was strong and well. On December 16 the infant did not seem well, and on the following day, after having passed a restless night, bronchial respiration and râles were detected at the base of the right lung. The temperature on that day varied from 38.3° C. (101° F.) in the morning to 39.4° C. (103° F.) in the evening. There were no symptoms except a slight cough, and the respirations were accompanied by an expiratory moan. Until December 23 it took about a quart of milk in the twenty-four hours, but on the 24th it refused to take any food, and the temperature, which had been gradually coming down so as to reach almost 38.3° C. (101° F.) in the evening, began to rise, and dulness and fine râles were detected in the left lower back. The pulse at this time rose to 160, but was regular and strong; the respirations varied from 60 to 70. The *alæ nasi* showed more active dilatation, and there was slight twitching of the arms and hands. The cough became more frequent, and there was slight diarrhœa. These symptoms continued for several weeks, when they lessened in severity and the temperature fell to the normal.

The case was treated with brandy and digitalis, and finally recovered entirely.

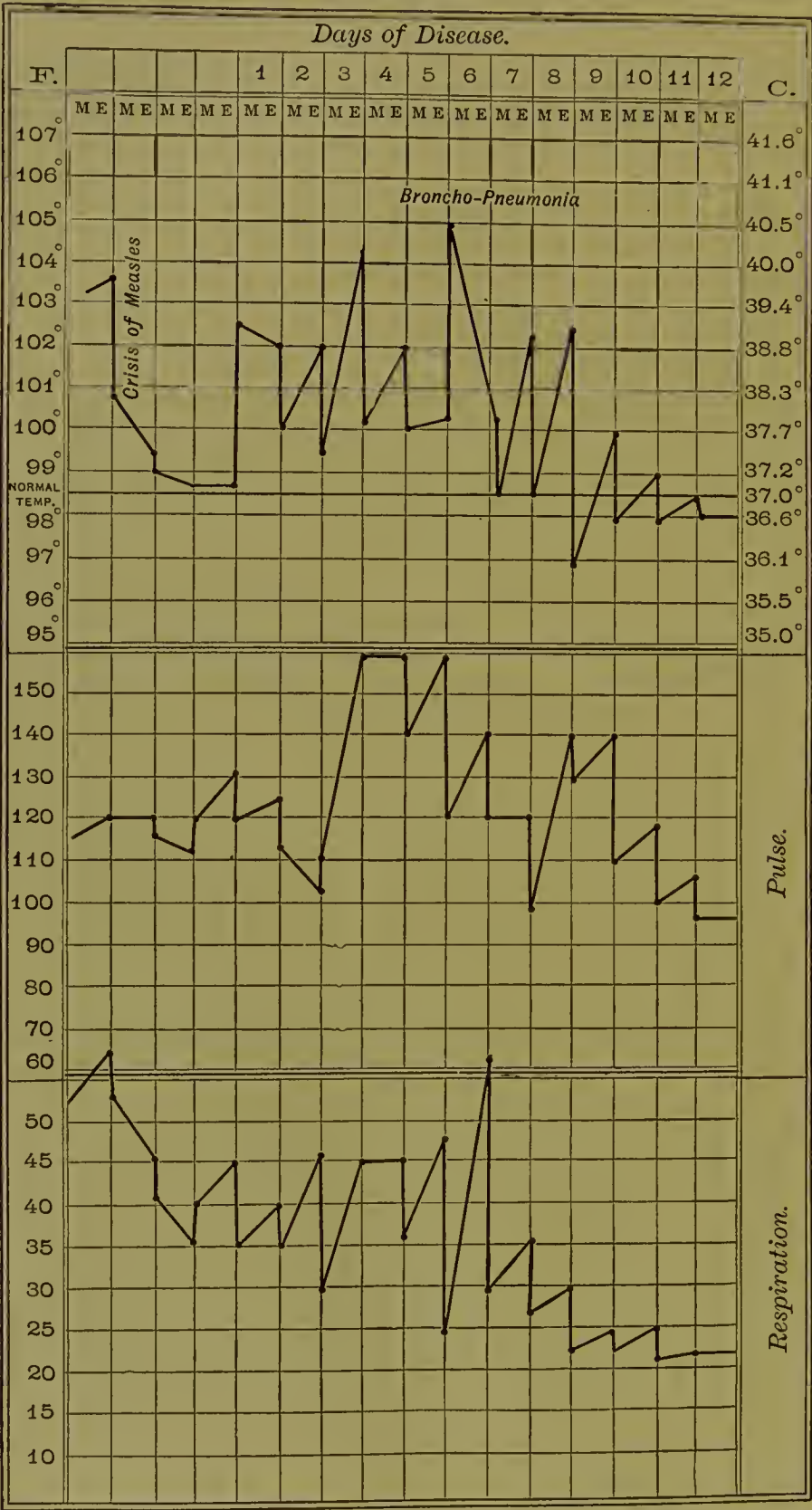
When this child was eight years old he was attacked with purpura rheumatica, followed in a month by nephritis, and one month later by a severe attack of broncho-pneumonia, which involved extensive areas in both lungs and was accompanied by constant



diarrhœa, delirium, cyanosis, and dyspnœa, resulting in death from exhaustion in the second week of the disease.

This next case, a girl, two years old (Case 464), had up to the present attack been strong

CHART 35.



Broncho-pneumonia following crisis of measles. Child, 2 years old.

and well. There is no tuberculous history in the family. She entered the hospital six days ago, and was placed in the isolating ward, as she was found to have measles. On entrance

her temperature was  $39.8^{\circ}\text{C}$ . ( $103.7^{\circ}\text{F}$ .), the pulse was 120, and the respirations were 62. An examination of the chest showed the heart to be normal. The percussion showed normal resonance and harsh puerile respiration, with numerous fine and coarse moist râles throughout both lungs. Nothing else abnormal was detected.

Five days ago the temperature fell to  $38.1^{\circ}\text{C}$ . ( $100.7^{\circ}\text{F}$ .), four days ago to  $37.3^{\circ}\text{C}$ . ( $99.2^{\circ}\text{F}$ .), and day before yesterday was just above normal. During these days the efflorescence rapidly faded, and she seemed better, though she occasionally had a harsh cough. Yesterday she had a slight rise of temperature, but there were no marked symptoms until to-day, when the temperature rose to  $40^{\circ}\text{C}$ . ( $104^{\circ}\text{F}$ .), the pulse to 160, and the respirations to 50. She is, as you see, very restless, and has considerable dyspnoea. She is pallid and sometimes slightly cyanotic. On physical examination there is found diminished resonance over an area in the lower part of the left back. Over this area the breathing is bronchial, and there is also a number of moist râles. On the right side of the thorax, especially at the base of the lung, there are numerous coarse moist râles and harsh respiration, but no dulness.

This case illustrates the rapid development of a broncho-pneumonia during an attack of measles, occurring after the temperature produced by the measles had fallen to the normal and while the efflorescence was disappearing. The physical signs show the presence of small areas of consolidation in the left lung, and the usual diffuse bronchitis throughout the right lung and parts of the left lung.

(Subsequent history.) This chart (Chart 35) shows the course of the temperature, pulse, and respiration during the next twelve days. The pulse continued to be rapid and the respirations to be somewhat raised for some days after the temperature became normal. The abnormal signs in the chest disappeared, and the child made a rapid recovery.

**Chronic Broncho-Pneumonia.**—I have already described the pathological lesions which occur in chronic broncho-pneumonia. In a certain number of cases, after a child has had an attack of acute broncho-pneumonia the physical signs of consolidation may persist, although apparent recovery has occurred so far as the general symptoms are concerned. When this occurs the fever may return after a variable period, and the child, after having become still more emaciated, may die after a number of months of exhaustion. Instead of this fatal issue, the child, as has been shown by Delafield, may be left with a chronic form of the disease, which may last for many years and be accompanied by symptoms of cough, dyspnoea, and at times periods of fever. The most common termination of these cases is in acute general miliary tuberculosis. In certain cases, however, where only a small portion of the lung has been affected, the child may recover as it grows older. Broncho-pneumonia of a subacute or a chronic type is so apt to develop in the lungs of young children during the course of any disease of a prolonged nature, that frequent examinations of the lungs should be made, in order that the insidious development of these pulmonary lesions may not be overlooked.

The treatment of these chronic cases of broncho-pneumonia is essentially climatic. The child should be taken to a warm dry climate of high altitude, where it can live in the open air, and where it will not be subjected to frequent atmospheric changes.

**ATELECTASIS.**—Atelectasis is a collapsed and unaerated condition of the air-vesicles. It may be congenital or acquired.

*Congenital atelectasis* arises because the infant has not sufficient general



vitality and respiratory power at birth to inflate fully all parts of its lungs. There may be an obstruction by mucus. There are in these cases areas of uninflated pulmonary vesicles of varying extent. These vesicles at the post-mortem examination can easily be artificially distended, and then cannot be distinguished from those which have been normally inflated.

The symptoms of congenital atelectasis are cyanosis, dyspnoea, rapid respiration, rapid, feeble, and often intermittent pulse, a temperature usually lowered, and dulness on percussion with lessened respiration over the atelectatic area. These are the typical physical signs of atelectasis, but in many cases some or all of these signs are absent and the areas of atelectasis are detected only at the post-mortem examination.

The prognosis in these cases varies according to the extent of the pulmonary tissue involved and the vitality of the infant. As a rule, the prognosis is very unfavorable.

The treatment of atelectasis is to stimulate the infant, and to endeavor to raise its temperature by means of a warm pack. In a number of cases I have found the administration of small quantities of oxygen to be of benefit. Artificial inflation of the air-vesicles has not proved to be an especially valuable form of treatment.

*Acquired atelectasis* is a symptom of some other disease, and I have already spoken of it sufficiently in connection with what I have said concerning broncho-pneumonia. Acquired atelectasis undetected during life is frequently found at the post-mortem examination of infants and young children dying of almost any disease.

**LOBAR PNEUMONIA.**—Lobar pneumonia is an acute self-limited disease of the lung, running a definite course and caused by the *diplococcus pneumoniae*.

FIG. 140.



Diplococcus pneumoniae.

**ETIOLOGY.**—Although lobar pneumonia may occur at any age, it is not met with so commonly in infancy and in early life as broncho-pneumonia. Exposure to cold, and especially to sudden atmospheric changes, apparently renders the individual more susceptible to the invasion of the micro-organism which causes this disease. Here is a specimen (Fig. 140) of this organism, taken from the sputum of a case of lobar pneumonia. It shows the morphology.

According to Delafield and Prudden, these germs during their development are distinctly spheroidal, but in their mature condition they often become slightly elongated and a little broader at one end than at the other, which gives them a lanceolate form. They are very apt to occur in pairs, and are frequently seen in short chains, but rarely in long chains. Very frequently when growing in the living animal the pneumococcus is surrounded by a distinct homogeneous capsule of varying thickness. The coccus itself is readily stained; the capsule is stained with difficulty.

**PATHOLOGY.**—The pathological condition which occurs in acute lobar pneumonia is an acute exudative inflammation which involves progressively the whole of one lobe, or the larger part of one lung, or portions of both lungs. There is no especial distinction between the lesions of lobar pneumonia as they occur in children and those which are met with in adults, except so far as the anatomical conditions differ according to the age of the individual. The stages of congestion, red hepatization, gray hepatization, and resolution take place in succession in the pneumonia of the child as in that of the adult. In the stage of congestion the lung is hyperæmic and œdematous and the air-vesicles contain fibrin, pus, granular matter, red blood-cells, and epithelial cells. The epithelium of the air-vesicles is swollen, and there are large numbers of white blood-cells in the capillaries. The large bronchi are congested. The small bronchi contain the same inflammatory products as do the air-vesicles. This stage lasts only a few hours, as a rule, but may be protracted for several days. When the exudation of the inflammatory products has reached its full development the presence of these products within the air-vesicles and bronchi causes the lung to be slightly enlarged, and at this time it is said to be in the condition of red hepatization. After the air-vesicles have become completely filled with exudation there follows a period during which the exudation first becomes decolorized and then degenerated. This is the period of gray hepatization. This happens at a variable time, which is usually shorter in children than in adults. The color finally becomes gray. The exudate then undergoes still further degeneration and softening, and is removed by the lymphatics. This is the stage of resolution. Resolution should begin immediately after defervescence and be completed within a few days, but it may not begin until a number of days after defervescence, and may be unusually protracted.

The bronchi are almost always affected in lobar pneumonia. The pneumonic process may occur in small patches, but usually involves an entire lobe. The lower lobes are the ones which are most frequently affected in early life, but the locality of the pneumonia is of pathological rather than of clinical importance, as the disease may attack any part of the lungs. It is generally a unilateral disease, but in some cases it may be bilateral.

**SYMPTOMS.**—The onset of acute lobar pneumonia is, as a rule, sudden, and in the infant or young child is frequently accompanied by vomiting and sometimes by convulsions; the latter, however, is uncommon as an initial symptom after the period of infancy. An initial rigor is uncommon. Pain



is probably present, but cannot usually be located by the child with the same precision as by the adult, young children often referring the pain to the abdomen. Cough is a common symptom, not only in the beginning of the disease but also during its whole course, and often seems to be painful. It may, however, be absent for several days in the beginning of the attack. There is rarely any expectoration before the seventh or eighth year. In some cases during the height of the disease there is delirium. In the milder cases the delirium may be merely a slight wandering, but in the more severe cases the children may become much excited, and the delirium may be accompanied by contracted or dilated pupils, and even involuntary passages of urine and of fæces, with continual movement of the head, muscular twitchings, and other symptoms which may simulate closely those of cerebro-spinal meningitis. In place of the delirium and the excited condition there may be a condition of stupor which sometimes simulates the stupor of tubercular meningitis. In another set of cases the nervous symptoms markedly simulate those of the non-tubercular form of meningitis. Meningitis in any form, however, rarely occurs in the course of pneumonia. Violence of the symptoms is not common. Marked cerebral symptoms seem to depend more on the height of the temperature and the extent of the lung involved than on any especial part of the lung being affected, such as the apices. In infancy and in the early years of childhood, in place of these cerebral symptoms there may be simply an apathetic condition during the height of the disease, and the infant, although somewhat somnolent and restless, often shows no other nervous excitement. The course of the disease is usually shorter in young than in older children.

The rate of both the pulse and the respiration is increased, but the greatest increase is shown in the rate of the respiration. This is much higher *proportionately* to that of the pulse than is usually the case in other affections which would be likely to simulate lobar pneumonia. There is commonly dilatation of the alæ nasi, and it is seldom that this symptom is absent in pneumonia. The pulse varies from many causes, among which is the nervous condition of the especial child. It may be 120 to 150.

The temperature is a very important symptom in acute lobar pneumonia, and is almost diagnostic of the disease. In the initial stage it rises at once to 39.4° or 40° C. (103° or 104° F.); it remains high, with slight remissions of about two degrees in the morning, for a number of days, and then in a large number of cases falls to the normal within twenty-four hours by crisis. The time when the temperature falls and the crisis takes place varies. It may occur as early as the third or fourth day, but is usually between the fifth and the eighth day. It may, however, be delayed until the ninth or tenth day, and in rare cases still longer. When the temperature falls at the crisis of the disease it is very apt to be subnormal, and to remain so for a number of days. Sometimes after the temperature has fallen to the normal it may rise again, but, as a rule, another rise of temperature points towards the involvement of some fresh area of the lung or to some complication,

such as pleurisy. The fall of temperature at the time of the crisis is often accompanied by symptoms of great prostration and even collapse, and it is therefore important in young children to watch carefully for the crisis and to be prepared to combat these symptoms. The normal height of the temperature in acute lobar pneumonia, according to the extensive observations of Holt, is from  $40^{\circ}$  to  $40.5^{\circ}$  C. ( $104^{\circ}$  to  $105^{\circ}$  F.). In children over three years of age the temperature curve resembles the adult type in being regular and falling by crisis, while under three years of age the proportion of typical cases is much less, and there is more irregularity in the course of the temperature, which may fall by lysis. The younger the individual the more likelihood there is to be a wide fluctuation in the range of the temperature, which has a tendency to be of the remittent type even in uncomplicated cases.

The physical signs of lobar pneumonia are the same as occur in adults. There is dulness on percussion over the affected area of the lung where consolidation has taken place, with bronchial respiration, increased vocal fremitus, and increased vocal resonance. In the initial stage of the disease fine râles are heard at times, but not so commonly in children as in adults. When resolution is taking place, moist râles of all sizes are heard. These are the typical signs of lobar pneumonia. In some cases the physical signs are entirely absent for a number of days, and the diagnosis has to rest upon the heightened temperature, the increased respirations and pulse, and the dilatation of the *alæ nasi*. In the early days of the disease the cough may be absent; this renders the diagnosis still more difficult. The cough may continue and the physical signs remain unchanged for a number of days after the temperature has fallen. The physical signs in some cases immediately disappear when the temperature becomes normal. Fine dry râles are not heard so frequently in the lobar pneumonia of young children as in that of adults.

In certain cases, where hepatization of the lung has taken place in the usual way and the crisis has come with a fall of temperature, resolution will fail to take place and the lung will remain solidified sometimes for a long period. Although an infection by the bacillus tuberculosis may be suspected in many of these cases, from their protracted course and from the prostration which usually accompanies them, yet such infection does not necessarily take place, and resolution often finally occurs. In these cases the lung is left apparently in the same normal condition as if this variation in the resolution had not taken place.

As an illustration of delayed resolution in lobar pneumonia I shall report to you the case of a little girl (Case 465), four years old, who, when she was perfectly well and strong, was suddenly attacked with vomiting, pain in the right side, and cough accompanied, according to her mother, by a reddish-brown sputum. Physical examination on the following day revealed nothing abnormal except a few fine moist râles at the base of the right lung behind. The pulse was 170, the respirations 60, and the temperature  $39.4^{\circ}$  C. ( $103^{\circ}$  F.). On the following day the temperature still remained raised, and there was dulness on percussion over the lower right lobe behind, with bronchial respiration. On the following day the dul-



ness had extended over the whole of the right lung in front and behind. The temperature varied from 38.8° to 39.4° C. (102° to 103° F.), the pulse from 150 to 160, and the respirations from 50 to 60. These symptoms continued until the eighth day from the onset of the attack, when the temperature was found to be 38.4° C. (101.2° F.), the respirations 48, and the pulse 160. During the next nine days the temperature, pulse, and respirations remained the same, and there was no change in the physical signs of the lung, except that in addition to the dulness and bronchial respiration a number of fine moist râles were heard in the back and in the axillary regions. The child was seen at this time by me in consultation with Dr. Calvin Ellis, and the physical signs were verified. During the next week no change took place in the temperature, pulse, respiration, or physical signs. Some days later the temperature fell to the normal, the respirations to 36, the pulse to 135, the dulness began to disappear, and the numerous coarse and fine moist râles of resolution appeared. Resolution took place rapidly, and a week later, thirty days from the onset of the attack, the lung appeared to be in a perfectly normal condition. From that time the child gained rapidly in strength and weight and recovered completely.

In some cases the child may show the rational signs of pneumonia, quick respirations, rapid pulse, dilatation of the alæ nasi, apathy, delirium, and perhaps vomiting and convulsions, for many days before the physical signs appear in the lung. In order to illustrate this delay in the appearance of the physical signs of lobar pneumonia I shall report to you briefly two cases which I saw in consultation with Dr. Chase, of Dedham. The whole course of the disease and the physical signs were so similar in both instances that one description will suffice for both.

They were two boys (Cases 466 and 467), brothers, the older boy being three years old and the younger sixteen months old. The older boy was attacked on November 19, and the younger one on November 20, with continuous vomiting, which lasted without much intermission until November 26. In addition to the vomiting the temperature rose in the first twenty-four hours to 40.5° C. (105° F.), and until November 26 varied from 40° to 40.5° C. (104° to 105° F.). The respirations varied from 40 to 50, and the pulse from 150 to 160. Both children soon became unconscious, were very restless, rolled their heads continuously, and had contracted pupils. On November 26 the temperature fell to 39.4° C. (103° F.), and during the next two days varied from 39.4° to 40° C. (103° to 104° F.). On November 27 a small area of absolute dulness with bronchial respiration was detected in the older boy over the left upper lobe in front, and on the following day in the younger boy over the left lower lobe behind. After the first few days there was slight cough in both cases, with movement of the alæ nasi. On November 30 the temperature in both children rose to 40.5° C. (105° F.), and until December 2 it varied from 40° to 40.5° C. (104° to 105° F.). On the evening of December 2 the temperature in the older boy suddenly fell from 40.5° C. (105° F.) to 35.5° C. (96° F.). The child became cold, the pulse became feeble, and the respiration could scarcely be detected. The application of the hot pack and an enema of hot brandy-and-water rapidly revived the child. The same fall of temperature occurred in the other boy on the following morning. In both children signs of resolution were detected before the temperature fell, the lungs in both cases rapidly became normal, and after a short convalescence the children recovered completely.

In some cases lobar pneumonia may attack both lungs. Again, after the disease has run its course and the temperature has fallen to the normal, a fresh portion of the lung may be attacked and the temperature may rise again. In rare instances in otherwise typical cases of lobar pneumonia I have been unable to detect any râles over the area of solidification throughout the whole course of the disease.

**DIAGNOSIS.**—The diagnosis of lobar pneumonia, when the typical temperature and the characteristic physical signs are present, is not difficult, but there are a number of atypical cases in which a doubt might easily arise for a number of days after the invasion of the disease. An early diagnosis from a pleuritic effusion and from other pulmonary affections is at times impossible.

The differential diagnosis between lobar pneumonia and a pleuritic effusion may be quite difficult in the early stages before the characteristic areas of dulness have been established. In both diseases dulness over a limited area, and bronchial respiration without any especial difference in the vocal fremitus and vocal resonance, and without evidence of a friction-rub or of râles, may make the two diseases simulate each other closely and compel us to wait for further developments before determining which disease is present.

From tubercular disease of the lung the differential diagnosis is usually not difficult, except in young infants, in whom the tubercular process with its corresponding symptoms may in rare cases simulate lobar pneumonia.

The disease from which a differential diagnosis should especially be made is broncho-pneumonia. Lobar pneumonia and broncho-pneumonia are so distinct, however, in their previous history, initial stage, course, and duration that, if care be taken to note closely all these stages of the two diseases and to arrive at a diagnosis from the evidence given by all the stages and not by any one stage, the diagnosis can, except in the very early days of the disease, usually be determined. Lobar pneumonia, in contradistinction from broncho-pneumonia, is a primary disease, characterized by a sudden onset and a regular temperature, the rise being sudden. This is accompanied by a corresponding rapidity of the pulse and respirations, dulness on percussion usually involving and limited to one lobe or one lung, with increased vocal fremitus and resonance, and bronchial respiration over the dull area. This is followed by a fall of temperature and by a rapid resolution. The duration is short and definite. Broncho-pneumonia, on the other hand, is usually secondary to a preceding bronchitis, occurring either alone or in the course of some other disease. It is characterized by a slow and insidious onset, except when occurring in the course of measles; it has an irregular temperature, the rise usually not being so sudden or so high as in lobar pneumonia, and the respirations and pulse slowly rising with the temperature. There is often an absence of change in percussion, the dulness if present showing itself in small patches and commonly in both lungs. There is also often an absence of marked vocal fremitus or vocal resonance, and of bronchial respiration, except where the patches of dulness are pronounced. Moist râles of all sizes may be heard in circumscribed areas throughout both lungs. The temperature is usually of a remittent type, and this condition lasts for weeks rather than days. The resolution is slow. The duration is often prolonged. If these pictures of the two diseases are borne in mind, an error in the differential diagnosis will seldom be made. In the doubt-



ful cases, where the characteristic course of either disease is absent, it will usually be found that we are dealing with a case of broncho-pneumonia, which is an exceedingly variable disease, rather than with lobar pneumonia, in which some of the characteristic features of the disease are almost invariably present.

In making the diagnosis between pneumonia and meningitis it is of much aid to remember that the *slow* intermittent pulse, slow irregular respiration, and moderate temperature of meningitis are uncommon in lobar pneumonia, where in most cases the pulse is quick and regular, the respirations rapid, and the temperature high. It is not uncommon, however, to find irregularities and intermissions in the *rapid* pulse of pneumonia. The younger the individual, as I have already stated in my lecture on meningitis (page 612), the more likely are the symptoms of tubercular meningitis to be replaced by those of the non-tubercular form of the disease, which may often simulate closely the symptoms of pneumonia. The convulsions which occur in pneumonia do not differ from those which occur in meningitis or, in fact, in any other acute disease. A careful physical examination should be made at every visit, once or twice a day if possible, as in this way the masked symptoms of a pneumonia may at times be detected where they would be overlooked if only an occasional examination were made.

After the first four or five days, as a rule, the differential diagnosis between cerebral disease and pneumonia is not difficult.

COMPLICATIONS.—The complications of acute lobar pneumonia are not very numerous. At times a pericarditis may occur, with its resulting effusion, but these cases are rare. The most common complication is a pleuritic effusion, which, especially in young infants, is apt to be purulent. In many cases the onset of the disease and its initial symptoms are apparently characteristic of pneumonia, and yet a few days later it becomes evident that a purulent pleuritic effusion has either complicated the pneumonia or was the original disease, simulating in its symptoms the early stage of lobar pneumonia. Lobar pneumonia is at times a serious complication of other diseases, and adds materially to their gravity.

GANGRENE.—One of the rare complications of lobar pneumonia is gangrene of the lung. This lesion is never found as a primary disease, and is rare in children. It is usually met with in weak, debilitated children whose circulation is impaired.

PROGNOSIS.—The prognosis of lobar pneumonia is very favorable. In young infants, or in those who are weak and debilitated, it is often fatal, but in comparison with broncho-pneumonia the percentage of recovery is very high. When the temperature rises to  $41.1^{\circ}$  C. ( $106^{\circ}$  F.) the prognosis is usually grave. The convulsions which occur in the initial stage of the disease in infants are commonly not of grave import. Occurring late in the disease they make the prognosis very unfavorable. When delirium occurs, although it may be severe, it does not render the prognosis especially un-

favorable. The fulminant type of the disease which sometimes occurs is a very fatal form.

TREATMENT.—As lobar pneumonia is a self-limited disease of short duration, the children are not so apt to die of exhaustion, and as a rule only an expectant treatment is called for. Where the disease occurs in very young infants it is safer to administer stimulants from the beginning. In children, however, it is often not necessary to use any drug whatever, and it is safer to wait until there are indications that the disease will not run a benign course before using drugs. Such indications are especially given by the temperature. Although at times a high temperature does not necessarily indicate danger, since a temperature of from  $40^{\circ}$  to  $40.5^{\circ}$  C. ( $104^{\circ}$  to  $105^{\circ}$  F.) is part of the regular course of the disease, yet if the temperature rises above this point it is well to reduce it by means of bathing and to give stimulants in the form of brandy. The child should be placed in a room of an equable temperature of  $20^{\circ}$  or  $21.1^{\circ}$  C. ( $68^{\circ}$  or  $70^{\circ}$  F.), and should be given milk every two hours. There is no necessity for making any external applications to the chest. The use of poultices is to be deprecated, and in my experience is usually without benefit except in certain instances for the relief of pain. The nursing is of especial importance, and close watchfulness, especially at the time when the crisis is expected to take place. At this time the temperature in infants and young children may fall with such rapidity to several degrees below the normal point that collapse often takes place, the skin is cold and moist, and sometimes the child becomes unconscious. Under these circumstances the pulse is feeble and intermittent, and in some cases death may occur unless active measures are taken for establishing reaction. The nurse should therefore be warned as the time for the expected crisis approaches to watch the child both night and day, and to have remedies ready to be used in case serious symptoms should arise. These remedies should be the external application of heat by means of the hot pack, and the administration of brandy by the mouth if the child can swallow, otherwise by rectal injection. I have known of a case (Case 468) where a child died in the collapse following the crisis of a lobar pneumonia. During the convalescence from pneumonia the child should be protected from atmospheric changes, cold, and dampness for some time.

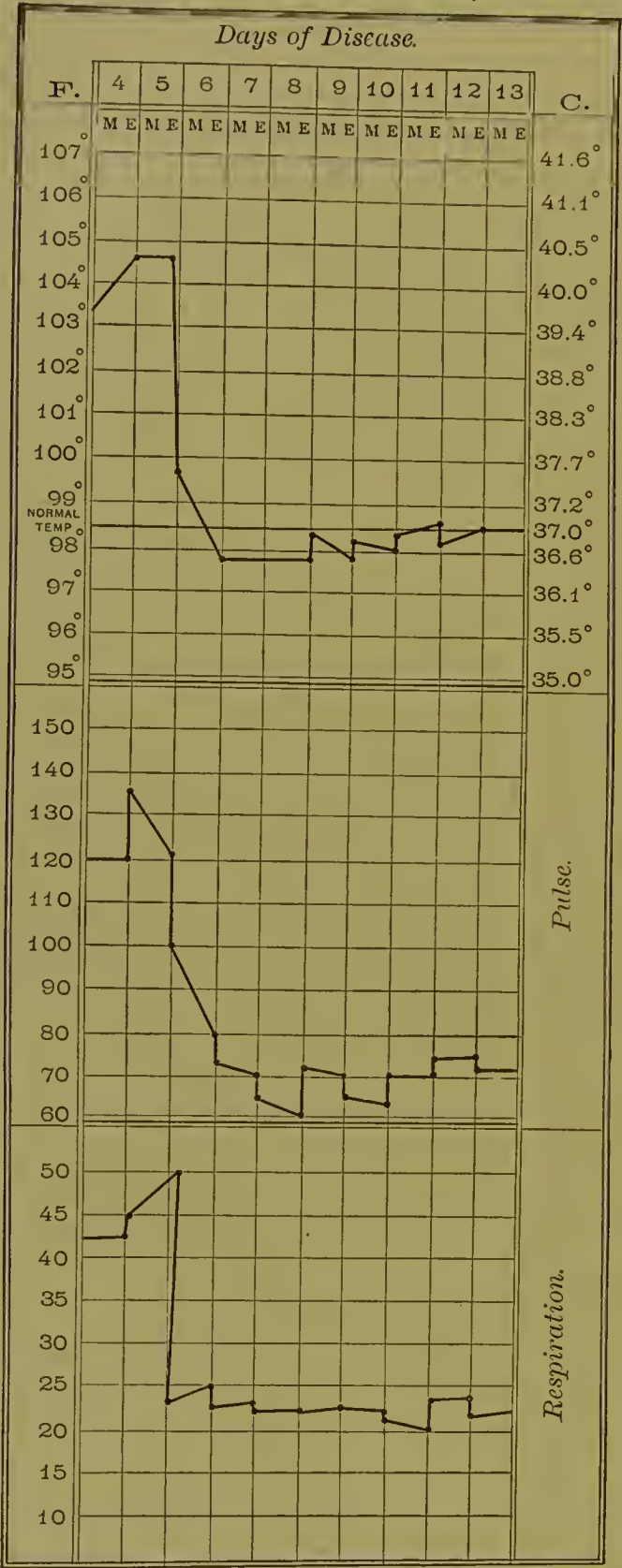
Lobar pneumonia may occur in the earliest days of life. I have met with a case (Case 469) which on the third day of its life developed a lobar pneumonia and died in twenty-four hours. The autopsy was made by Dr. W. F. Whitney, and the characteristic hepatization was found.

Here is a boy (Case 470), eight years old, who entered the hospital on the fourth day of an attack of lobar pneumonia. The attack began with vomiting and cough, but no pain, expectoration, or chill. An examination showed the right lung to be normal. On the left side of the chest an area corresponding to the lower lobe in the back was found to show absolute dulness on percussion, bronchial respiration, and many fine moist râles. Nothing else abnormal was detected. The child was very restless, but on the following day, the fifth



from the onset of the disease, the temperature fell by crisis to the normal point. This chart (Chart 36) shows the typical temperature, pulse, and respiration of a case of lobar pneumonia.

CHART 36. (CASE 470.)

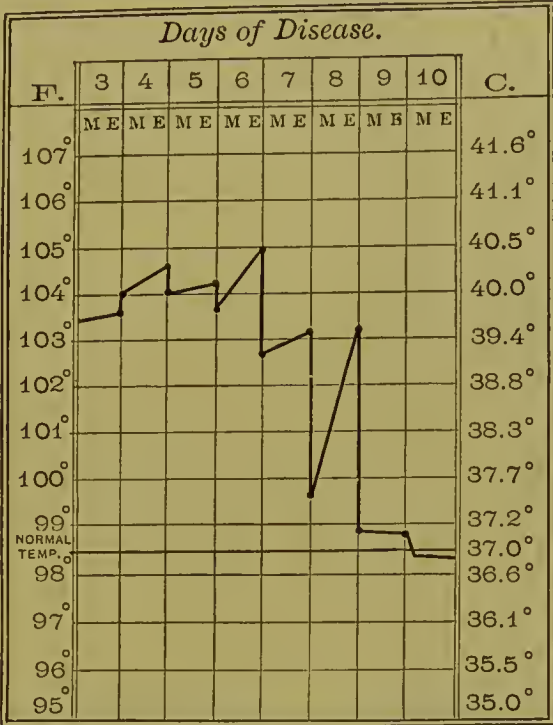


Lobar pneumonia    Male, 8 years old.    Crisis on fifth day of disease.

(Subsequent history.) After the crisis the child improved rapidly, and the physical signs disappeared in ten days.

This little girl (Case 471), two and a half years old, entered the hospital on the third day of an attack of lobar pneumonia. To-day is the tenth day from the onset of the disease, and this chart (Chart 37) shows one of the variations in the crisis which is quite frequently met with in young children.

CHART 37.



Lobar pneumonia. Irregular crisis on eighth day. Female, 2½ years old.

In this case the consolidated portion of the lung was the left lower lobe. The resolution was rapid and convalescence normal.

This little girl (Case 472), eight years old, had pertussis when she was fourteen months

CASE 472.

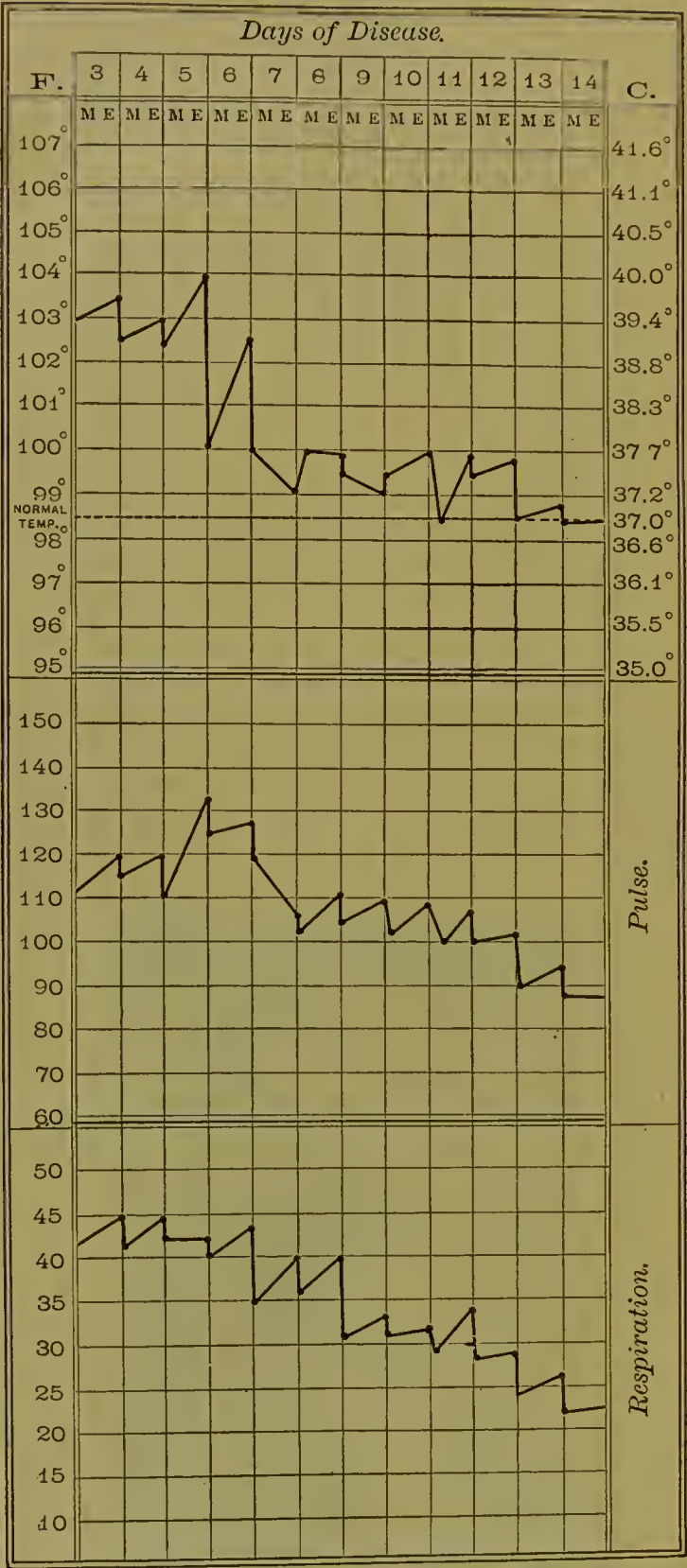


Lobar pneumonia. Female, 8 years old. The part of the lung involved by the pneumonic process is shown by black lines, and the area of diminished resonance and the fine râles are marked by black spots.



old, scarlet fever when she was five years old, and measles when she was six years old. Five days ago she lost her appetite, was very feverish, and was attacked with acute pain

CHART 38. (CASE 472.)



Lobar pneumonia. Female, 8 years old. Irregular crisis on sixth day.

referred to the left side of the epigastrium and the lower part of the left axillary region. She has since had a haeeking paroxysmal cough, with no expectoration. For the past few days she has been delirious. She vomited twice yesterday, and is very weak. Her tongue,

as you see, is heavily coated, the *alae nasi* are working, her face is deeply flushed, and she has dyspnoea to such an extent that she has to be propped up on pillows.

Her respirations are 45, difficult and painful, her pulse 120, and her temperature  $39.5^{\circ}$  C. ( $103.2^{\circ}$  F.). A physical examination detects nothing abnormal in the front of the chest or in the right back. There is absolute dulness in the left back, beginning at the fifth rib and extending to the base of the lung and into the axillary region. Over this area of dulness there is increased vocal fremitus and bronchial respiration. In this area, also, there are a few moist râles. Just above the upper border of the area of absolute dulness there are diminished resonance and a number of fine râles. This is the fifth day of the disease. Although the general condition of the child seems to show no especial change, yet the physical signs show that resolution has begun and that we may at any time expect the crisis to occur.

(Subsequent history.) On the following day the temperature fell to  $37.7^{\circ}$  C. ( $100^{\circ}$  F.) in the morning, but rose again in the evening to  $39.1^{\circ}$  C. ( $102.5^{\circ}$  F.). On the following day, the seventh day from the beginning of the attack, the temperature fell to  $37.2^{\circ}$  C. ( $99^{\circ}$  F.), and then varied from  $37.7^{\circ}$  C. ( $100^{\circ}$  F.) to  $37.2^{\circ}$  C. ( $99^{\circ}$  F.) until the eleventh day, when it became normal. The chart (Chart 38) shows the pulse and respiration up to the fourteenth day from the beginning of the attack.

This case is one which illustrates the fact that the physical signs of resolution may sometimes appear before the temperature falls and the crisis comes; also that at the time of the crisis the temperature may fall, then rise again for twelve to twenty-four hours, and then fall to the normal, as in this case. The child recovered completely.

This boy (Case 473), six years old, was taken sick four days before entering the hospital.

#### CASE 473.



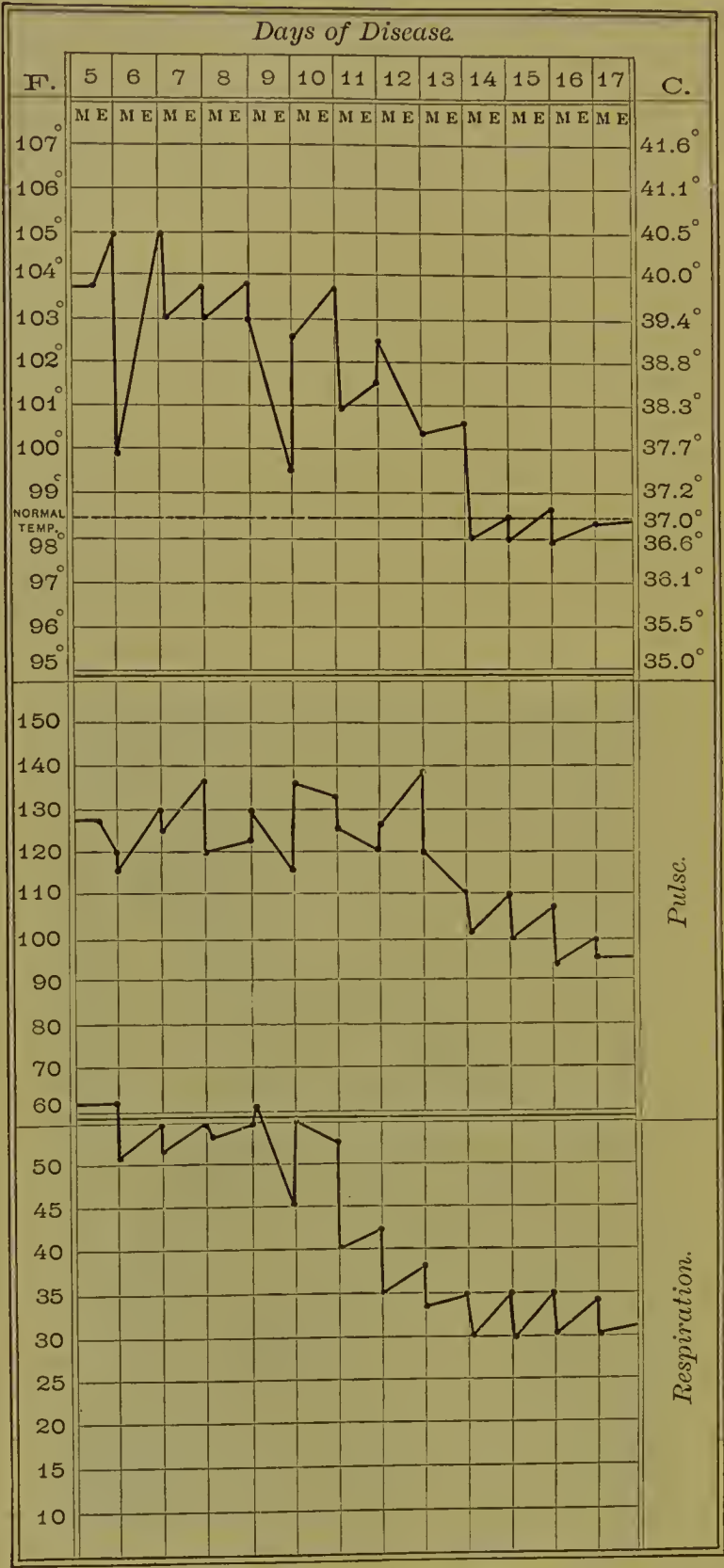
Lobar pneumonia. Three invasions. Male, 6 years old.

On entering the hospital his pulse was 128, his respirations 60, and his temperature  $39.8^{\circ}$  C. ( $103.8^{\circ}$  F.). A physical examination showed that there was absolute dulness over the entire upper lobe of the right lung. Over this area of dulness there were bronchial respiration and increased vocal resonance. There was also an occasional high-pitched



râle. The left lung was normal. I have marked the lower border of the dulness produced by the consolidated upper lobe by a black line extending from the sternum just above the

CHART 39. (CASE 473.)



Lobar pneumonia. Male, 6 years old.

right mamma and around into the axillary region. On the morning of the sixth day from the beginning of the attack the temperature fell to 37.7° C. (100° F.), but rose again in the evening to 40.5° C. (105° F.), and a physical examination then showed that the middle

lobe of the right lung was involved in front, as I have indicated by this second black line below the one which I have just described. The temperature during the next two days remained between 39.4° and 40° C. (103° to 104° F.), but on the following day, the ninth from the onset of the disease, the temperature suddenly fell to 37.6° C. (99.7° F.) in the evening, but rose the next morning to 39.3° C. (102.8° F.), and in the evening rose to 39.9° C. (103.8° F.). A physical examination then showed that the whole of the lower lobe was involved, as I have indicated by the third black line. On the following day the upper lobe began to show evidence of resolution, and the temperature fell to 38.3° C. (101° F.). Two days later the temperature began to fall by lysis, the physical signs of the upper and middle lobes entirely disappeared, and the temperature reached the normal point on the fourteenth day from the time of the onset. On the seventeenth day from the time of the onset the lower lobe was also found to be in a normal condition, and from that time convalescence was uninterrupted.

Here is the chart (Chart 39), which shows the temperature, pulse, and respirations in this case from the fifth to the seventeenth day of the disease.

In some rare cases the infection in lobar pneumonia is so intense that a rapidly fatal issue may occur.

I have seen a little girl (Case 474), nineteen months old, who had been having so mild an attack of diarrhœa that she was playing about out of doors, suddenly attacked in the afternoon with convulsions and a temperature of 40.5° C. (105° F.). The convulsions continued during the night, and she soon became comatose. On the following day the temperature still remained at 40.5° C. (105° F.), the respirations were much accelerated, and the pulse was about 120. An area of absolute dulness over the left lower lobe behind, with bronchial respiration and increased vocal resonance and fremitus, rapidly developed. The child did not respond to treatment, and died in the evening.

**TUBERCULOSIS OF THE LUNG.**—Tuberculosis of the lung is an affection in which certain lesions are produced in the lung by the bacillus tuberculosis. Although this tubercular affection may attack any organ or any part of the body, yet whenever it occurs elsewhere it is almost invariably found in the lung. It is well, therefore, to speak of this especial manifestation of tuberculosis in connection with diseases of the lung.

**ETIOLOGY.**—The cause of tuberculosis, as I have just stated, is an organism, the bacillus tuberculosis. Here is a specimen (Fig. 141, page 994) which shows the morphology of this organism.

These organisms are, according to Delafield and Prudden, slender, filamentous bacteria varying in length from one-quarter to one-half the diameter of a red blood-cell. They are frequently curved and bent, and may form short chains. This bacillus may retain its vitality for many weeks in a dried condition, but is killed by an exposure of fifteen minutes to a temperature of 100° C. (212° F.). In most cases it finds its way to the tissues by inspiration, although it may also gain access to the body by being swallowed.

**PATHOLOGY.**—The pathological conditions which result from infection by the bacillus tuberculosis are very numerous. The lesions in the child do not differ from those which occur in later life, and I shall therefore not describe them in detail.

The ordinary chronic tubercular lesions met with in adults are seldom



seen in children, and it is rare for the tubercular process in children to begin at the apices of the lungs and gradually extend downward, as is common in adults. When this occurs, it is usually in the later years of childhood, when the conditions are beginning to approximate those of later life. The most frequent entrance of the tubercular affection to the lung is through the bronchial glands. This fact has been especially studied and described

FIG. 141.



Tubercle-bacilli taken from the sputum in a case of tuberculosis of the lung.

by Northrup. According to this investigator, in most cases the infection of tuberculosis in children is effected by the entrance of the tubercle-bacilli into the respiratory passages with the inspired air, and the lodging of them in the mucus of the air-passages or the alveoli of the lungs. They may then pass through at any point, and, being taken into the lymph-spaces, traverse the lymph-canals to the nearest glands and be retained there. These glands at the base of the lung receive and filter everything brought to them from the bronchial tract. The subsequent career of the bacilli depends upon the power of the tissues to withstand their further progress. They may die, or may remain inactive for a long period and later develop a tubercular process in the glands. These tubercular glands may finally break down and thus allow the bacilli to penetrate different portions of the lungs and produce their characteristic lesions.

Tuberculosis of the lungs may occur in two forms: (1) *acute tubercular broncho-pneumonia*, and (2) *chronic tuberculosis of the lungs*.

**Acute Tubercular Broncho-Pneumonia.**—According to Osler, acute tubercular broncho-pneumonia is common in children from the sixth month to the fifth year, a large proportion of the cases, however, occurring after the second year. It is common in children who have been debilitated by previous illnesses, and occurs especially after measles, pertussis, scarlet fever, and diphtheria, being most frequent in the first two. It may, however, develop in perfectly healthy, well-nourished children, and also, as Osler has

expressed it, may be a terminal process in cases in which local tubercular disease exists in other parts, such as the skin, bones, lymph-glands, or the uro-genital tract.

As in the other forms of broncho-pneumonia, the initial lesion is a bronchitis and peribronchitis, the distinguishing tubercular features being caseation and necrosis of the consolidation with the presence of the tubercle-bacilli. The accompanying phenomena of atelectasis and emphysema occur as they do in non-tubercular broncho-pneumonia. In some cases the non-tubercular broncho-pneumonia precedes the tubercular disease, this occurring particularly after measles, scarlet fever, diphtheria, and pertussis. According to Mosny, where the tubercular broncho-pneumonia follows the non-tubercular form, in addition to the lesions of the latter disease, there are found true tubercular processes, such as peribronchial nodules, tubercular infiltration, and caseous areas. Where the patient is the subject of a latent tuberculosis, such as may follow one of the infectious diseases, a non-tubercular broncho-pneumonia may also develop. In these instances, according to Mosny, the lesions may be seen surrounding the tubercular peribronchitic nodules, or foci of non-tubercular or tubercular broncho-pneumonia are found scattered through the apices of the lung.

**SYMPTOMS.**—The symptoms of acute tubercular broncho-pneumonia are very similar to those of non-tubercular broncho-pneumonia. According to Osler, in most cases the onset of the disease simulates that of the ordinary non-tubercular broncho-pneumonia so closely that a differential diagnosis between the two diseases cannot be made until after death, and even then the post-mortem appearances may not be those distinctive of tubercular disease, and the pathological diagnosis can be determined only by finding the bacillus tuberculosis. The children may be attacked with cough, a heightened temperature, and the physical signs of broncho-pneumonia. The physical signs, as would naturally be expected, are usually found in the back and lower portion of the lung rather than at the apices, as in adults, on account of the usual nidus of the tubercular lesions,—namely, the bronchial lymph-glands. In some cases the onset of the disease is not so acute, and its course not so rapid. The child emaciates and has only a moderate fever, but later the development of such symptoms as sweating, chills, and hectic, together with the signs of softening and breaking down of the lung-tissue, leads us to suspect that we are dealing with tuberculosis of the lung.

**DIAGNOSIS.**—The diagnosis, as a rule, is to be made by taking into consideration the family history of the child, as the tissues of children whose parents are tubercular show an especial liability to infection by the bacillus tuberculosis. The diagnosis can be made positively only in those cases where a specimen of the sputum can be obtained and examined for the bacillus tuberculosis.

**PROGNOSIS.**—The prognosis is invariably unfavorable.

**TREATMENT.**—The treatment of tubercular broncho-pneumonia is the same as that of the non-tubercular form.



**Chronic Tuberculosis of the Lungs.**—Chronic tuberculosis of the lungs as it is ordinarily met with in adults is rarely seen in young children. During the first three months of life tubercular disease of any form is very rare, but in the latter part of the first year it becomes very common. The tubercular lesions which are found in the lungs in later life also occur in early life. Although cavities are not so commonly found in young children as in adults, it is not so much that they do not exist as that, their locality being more at the root and central portions of the lung, they are more difficult to detect on physical examination. It has been noticed that large cavities at the apex of the lung are rare in early life, but become more common as the child grows older. Tubercular disease of the lung is very irregular in the extension of its lesions in young children. Much more advanced lesions are usually found at the post-mortem examination than are detected during life. As I have already stated, the primary lesion of chronic tuberculosis of the lungs is commonly a tubercular broncho-pneumonia.

**SYMPTOMS.**—The symptoms of chronic tuberculosis of the lungs differ but little in the child from those seen in the adult, and are marked by the same irregularities in their course. This is due to the varied forms of the lesions. In young infants the symptoms are so often obscure and the physical signs of the serious pathological conditions which exist in the lungs are so frequently masked that the diagnosis is apt to be very doubtful. There is often a history of tuberculosis in the parents. The more common symptoms of chronic tuberculosis of the lungs are gradual loss in weight, strength, and appetite, irregular and moderate fever, hectic, and sweating. The physical signs are slowly increasing dulness in certain areas of the lung, especially in the back, accompanied by râles and other evidences of solidification. Later in the disease the characteristic signs of cavities may develop. Cough is usually present, though it is sometimes so slight in the beginning as not to be especially noticed by the parents. Hæmoptysis is rare in infants and in young children, but may be present in older children as they approach the age of puberty. As the disease progresses there is dyspnoea, usually of a moderate grade, with cyanosis, but in some cases considerable destruction may have taken place in the lung-tissue without the presence of any especial dyspnoea.

The course of chronic tuberculosis of the lungs is rather more rapid in children than in adults, and it is seldom that the long-protracted course of the disease so frequent in adults is met with in children. Sometimes, however, the child improves in its general health and may live for many years. In these cases the terminal phalanges of the fingers may become clubbed, and there is usually dyspnoea on exertion.

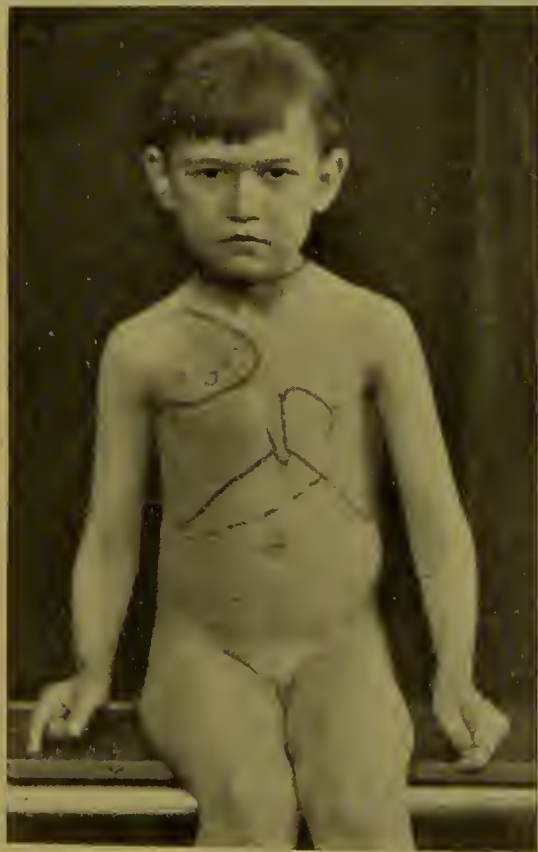
**DIAGNOSIS.**—The diagnosis is to be made from chronic empyema and from chronic non-tubercular broncho-pneumonia. The former disease can be readily eliminated by making an exploratory aspiration, but the latter can often be distinguished only by means of a bacteriological examination. In older children, where a specimen of the sputum can be obtained, the

diagnosis is readily made by the detection of the bacillus tuberculosis. In younger children, in whom expectoration does not take place, the diagnosis is much more difficult, but if the children are carefully watched it is often possible to obtain a specimen of the sputum if the child happens to vomit, in which case particles of sputum may be coughed up with the vomitus and can be separated from it and examined.

PROGNOSIS.—The prognosis of chronic tuberculosis of the lungs where the symptoms are at all advanced is very unfavorable, but the post-mortem examinations of so many individuals who have died of non-tubercular diseases show the presence of old tubercular lesions which have apparently ceased to be of grave import, that we must acknowledge that it is possible for many cases to survive the invasion of the bacillus tuberculosis.

TREATMENT.—The treatment of chronic tuberculosis of the lungs is essentially climatic, and the children should be removed at once, if possible, from a climate where the altitude is low and the atmosphere damp and subject to great variations. Too high altitudes are also to be avoided. Where the child cannot be removed to a more favorable climate, strict attention to its general hygiene and to its food will in some cases be followed by an apparent arrest of the tubercular process.

CASE 475.



Chronic tuberculosis of the lung. Female, 8 years old.

This little girl (Case 475), eight years old, has a history of tuberculosis in her family. She had an attack of pertussis when she was six years old, and some months ago an attack of measles. Following the attack of measles she began to have headache, cough, and ex-

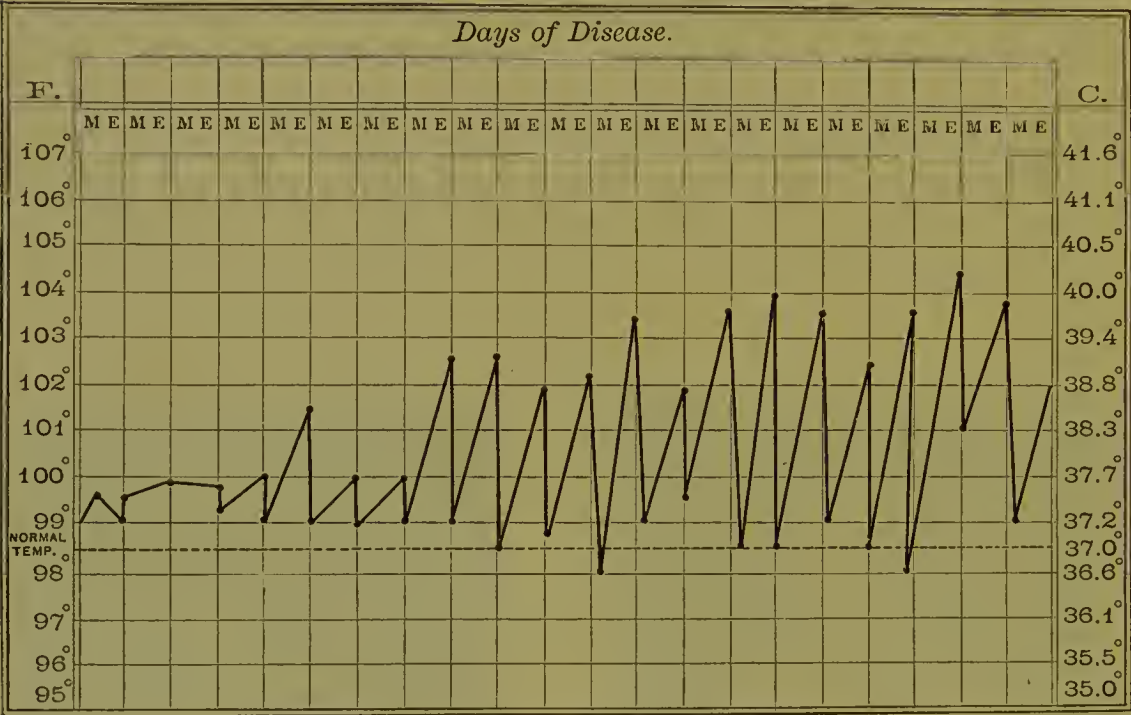


pectoration. She complained of pain in her chest and abdomen, and of chilly sensations, and has progressively lost in weight and strength. A physical examination shows the skin to be dry and harsh and the heart normal. The left lung in front appears to be normal. Behind over a small area at the upper part of the lung there are dulness, bronchovesicular breathing, and some fine moist râles.

Over the right upper lobe in front and behind there is dulness, and the expiration is prolonged and high-pitched. Over the dull region are heard medium and fine moist râles. I have indicated the borders of the areas of dulness by black lines, and the râles by black spots. I have also shown the area of cardiac dulness, the lower part of the sternum, and the lower border of the ribs, by dark lines, and the edge of the liver, which seems to be somewhat enlarged, by an interrupted line.

This chart (Chart 40, Case 475) shows the irregular temperature which is commonly seen in cases of chronic tuberculosis of the lungs and is of a remittent type.

CHART 40.



Chronic tuberculosis of the lung.

The expectoration has not yet been examined for the bacillus tuberculosis, but the diagnosis is not doubtful, as the child is gradually failing and the signs of disorganization of the lung are slowly progressing.

**PERTUSSIS (Whooping-Cough).**—Pertussis is a highly infectious disease affecting the respiratory tract and characterized by periods of spasmodic coughing, succeeded by a prolonged inspiration and accompanied by a peculiar sound called the “whoop.”

The cause of pertussis is probably a micro-organism, but this organism has not yet been determined. It is supposed that the contagium can be carried by a third individual, but usually it is directly communicated from one person to another. This contagium is probably contained in the expectoration, and in this way houses where the disease is present may become a source of infection. The disease commonly occurs in epidemics during winter and spring. Sporadic cases occasionally appear, and in large cities

the disease is often endemic. Pertussis seems to have some especial relation to measles, as children with the latter disease are liable to contract pertussis, and in like manner those with pertussis are liable to contract measles. Pertussis may occur at any age, and the disease has even been known to be contracted in utero. One attack usually protects from a second. Debilitated children with catarrh of the respiratory tract are more subject than others to the contagium of pertussis.

**PATHOLOGY.**—There are no pathological lesions distinctive of uncomplicated pertussis. The condition which characterizes the paroxysmal attacks is extreme congestion of the different organs, such as the meninges, the lungs, the heart, and the kidneys. In grave or fatal cases the lesions are those which arise either from mechanical accidents, as emphysema or hemorrhage in various parts, as the eye or the meninges, or from such complicating diseases as broncho-pneumonia with its accompanying bronchitis and atelectasis. The bronchial glands are often found to be enlarged.

**SYMPTOMS.**—The period of incubation of pertussis is variable, but is usually less than two weeks. The symptoms in the beginning, and often for several weeks, are simply those of a bronchial catarrh with a slight rise of temperature and a cough which, though sometimes spasmodic, is often indistinguishable from that of an ordinary bronchitis. After a period varying from a few days to two or three weeks, the cough becomes more severe and of a more decidedly spasmodic character, and the peculiar whoop which characterizes the disease appears. The cause of these paroxysms seems to be a spasm of the larynx. This is accompanied by a feeling of suffocation. The paroxysm begins with a number of short, spasmodic, expiratory coughs, succeeded by a long-drawn inspiration and by the peculiar whoop. During the paroxysm, especially in severe cases, the face and mucous membranes become cyanotic, the eyes protrude, the conjunctivæ are congested, and the child looks as though it would die of asphyxia. After a few seconds the child, with a convulsive cough, expels some tenacious mucus, and is then relieved, or the attack returns again, and again subsides, and the symptoms of asphyxia pass away. These paroxysms are often followed by vomiting. They may occur only four or five times in the twenty-four hours, or again much oftener,—at times thirty, forty, or fifty times. At the onset of the attack the children are usually very much frightened, and either run to the mother or nurse for aid, or go to some part of the room where they can be undisturbed during the attack. In certain children, after the severe paroxysms have lasted for some time, a small ulcer is formed on the frænum of the tongue. This is because the frænum is driven against the lower edge of the teeth during the paroxysms. During the course of the paroxysmal stage of pertussis it is quite common to have subconjunctival hemorrhages; rarely deeper-seated hemorrhages take place in the meninges and in the deeper parts of the eye. In protracted cases petechiæ sometimes appear in the skin. Epistaxis may also occur.

Examinations of the chest during the attack have shown that the pul-



mouary resonance is lessened during the expiratory stage and is clear during the prolonged inspiration. The auscultation usually shows diminution or absence of the respiratory murmur. Bronchial râles are heard occasionally.

Koplik has noticed an increase in the area of the relative cardiac dullness during the paroxysmal stage of pertussis, which is often accompanied by a slight blowing murmur limited to the apex of the heart. This may well occur from the engorged condition of the right side of the heart, which subjects the heart to a great strain and may thus result in dilatation. The heart-sounds are apt to be irregular during the paroxysm, and in protracted cases during the intervals the pulse is often irregular and quickened, while the respirations are not especially increased unless some complication has arisen. In severe cases of pertussis the kidneys are sometimes congested, as shown by the appearance in the urine of albumin, casts, and blood-cells. Sugar has also been found quite frequently.

After the disease has lasted for some weeks there is usually a certain amount of œdema of the face, especially under the eyes. The paroxysms are precipitated by nervous excitement or by an irritation in the throat or the respiratory tract, such as may result either from swallowing or from the inhalation of dust. The stage which is accompanied by the whoop and the more exaggerated paroxysms commonly lasts for three or four weeks, or even longer. The paroxysms then become less severe, and, although the cough continues, the whoop gradually becomes less frequent, and after three or four weeks more ceases entirely. When uncomplicated, the duration of the disease is usually three or four months. Slight changes in the atmosphere or exposure will give rise to a relapse. The relapses, however, are not, as a rule, of a severe type, and in these cases the cough seems to arise from renewed irritation of the sensitive mucous membrane of the respiratory tract rather than from a fresh infection by the specific germ. A persistent cough following an attack of pertussis may sometimes, according to Delafield, be caused by an insidious form of broncho-pneumonia.

The period of infection is supposed to last for a certain time after the whoop has ceased, and if the cough continues it is well to allow for a period of infection of three weeks after this cessation. It is possible, however, that the whoop may occasionally occur for long periods after the child has ceased to be a source of infection to other individuals.

COMPLICATIONS.—The complications which arise in pertussis are usually of a grave nature. The dangers from hemorrhages, unless in the form where they occur in the meninges, are not great. The complication of broncho-pneumonia is very serious, and often fatal. Severe and even fatal emphysema may occur in pertussis.

Convulsions may arise not infrequently in infants and may end fatally, in these cases usually being caused by general reflex disturbance, by cerebral congestion, or by some cerebral lesion. Spasm of the glottis may also very rarely cause death in greatly debilitated children. Excessive and

obstinate vomiting at times becomes a serious complication, and may reduce the child's strength to a point which often gives rise to a doubt as to its recovery. It is an especially grave complication in infants who are already much debilitated.

DIAGNOSIS.—The diagnosis of pertussis cannot, as a rule, be made until the child whoops. Sometimes, however, where another child in the family has undoubted pertussis, a spasmodic cough may allow the diagnosis to be made before the whoop has developed. It is probable that a child may have pertussis without at any time developing the whoop.

In some children a simple catarrhal laryngitis will simulate pertussis quite closely; but, although in these cases there are paroxysms of spasmodic coughing, a pronounced "whoop" does not occur, and the symptoms do not progressively increase and last for a long period. The diagnosis of pertussis can usually be made by the swollen aspect of the face, the paroxysmal cough followed by the expulsion of tough mucus and vomiting, and the long duration of the attack.

PROGNOSIS.—Pertussis is a very serious affection in young infants, and also in older children who are debilitated or poorly cared for. Where it is complicated it is one of the most fatal diseases which occur in early life. When it occurs in older children the prognosis is favorable, provided that they have previously been well and strong, that they are well cared for, and that no complications arise.

In some cases young infants, if their vitality is unusually good, and if they are carefully nursed and made to take a sufficient amount of food, show remarkable powers of resistance during attacks of pertussis.

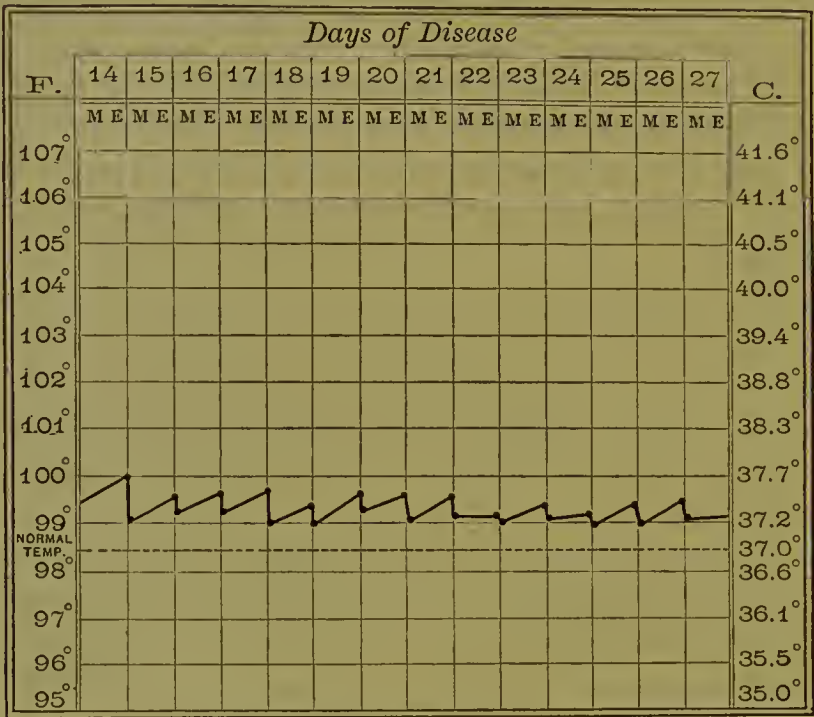
A case of this kind that came under my care was that of an infant (Case 476), five months old. In March she suffered from an attack of epidemic influenza, which lasted about twelve days, and from which she finally recovered. She was then attacked with measles, and after the temperature had fallen to the normal point she was attacked with pertussis. After two or three days the cough increased in severity, and after two weeks the infant began to whoop. The attack lasted for two months, and she finally recovered. During the whole course of the disease she took over 600 c.c. (20 ounces) of modified milk in the twenty-four hours, and for a short time small doses of brandy were given. No drugs were administered. Here is a chart (Chart 41, page 1002) which shows the average range of temperature for two weeks when the disease was at its height.

TREATMENT.—In the treatment of pertussis we must take into consideration the age of the individual, the stage of the disease, and the presence or absence of complications. In the early months of life, after the disease has lasted for a week or ten days and has become more severe, the infant will usually show symptoms of general circulatory disturbance. The great strain thrown upon the heart during the paroxysms quickly affects the general strength of the infant, a marked interference with its nutrition soon appears, it loses in weight, and often it refuses its food. At times it will become somewhat cyanotic even between the paroxysms, and there is danger not only from the severity of the paroxysm, but also from



the vitality of the infant being so much interfered with as to prevent its recovery. In cases of this kind the nursing is of the utmost importance. The infant should never be left alone, should always be taken up whenever a paroxysm is approaching, and should be assisted in various ways until the paroxysm is over. Holding the infant in different positions, sometimes bending the head and body forward at the end of the paroxysm so as to aid by gravity the expulsion of the tenacious mucus, is desirable. At times, also, the finger covered with a thin cotton cloth can be quickly introduced

CHART 41. (CASE 476.)



Pertussis. Female, 5 months old.

into the throat and the mucus withdrawn in this way. It is of the utmost importance that the infant should be surrounded continually by fresh air. For this purpose two rooms should be used, if possible, one of which should have all the windows thrown wide open, so that the air can be completely changed before the infant is brought into it, and the patient should be alternately taken from one room to the other, the temperature of the rooms being kept as equable as possible. The nutrition of the infant is so easily affected that the utmost attention should be paid to the administration of the food. Small quantities of a milk carefully modified to suit its digestion should be given at frequent intervals, preferably after the occurrence of a paroxysm, as it is then more likely to retain the milk in its stomach a sufficient length of time for it to be absorbed before the next attack. The amount of food which the infant retains in the twenty-four hours is an important factor in the treatment. In infants of from six to twelve months at least 600 to 750 c.c. (20 to 25 ounces) of milk should be taken and retained in the twenty-four hours. When the amount is lessened to 360 or 450 c.c. (12 to 15 ounces), the infant's nutrition, as a rule, suffers to such an

extent that unless this amount can be increased a fatal issue is likely to result.

Stimulants, in the form of brandy or whiskey, should be given early in the attack. Where the cyanosis is a prominent feature and the pulse is irregular and intermitting, small doses of digitalis should be given. In these cases, also, the administration of oxygen is a valuable adjunct to the treatment. At the height of the attack, when the paroxysms are severe and especially frequent at night, the burning of cresoline in the room at night is in some cases beneficial. In the milder cases not accompanied by the more severe symptoms which I have just enumerated, belladonna or atropine often proves valuable. There is no drug, however, which is a specific for pertussis at any age.

For older children whose health has previously been good, there is no especial treatment, except that they should have as much fresh air, free from dust, as possible, and that food should be given them after they have vomited.

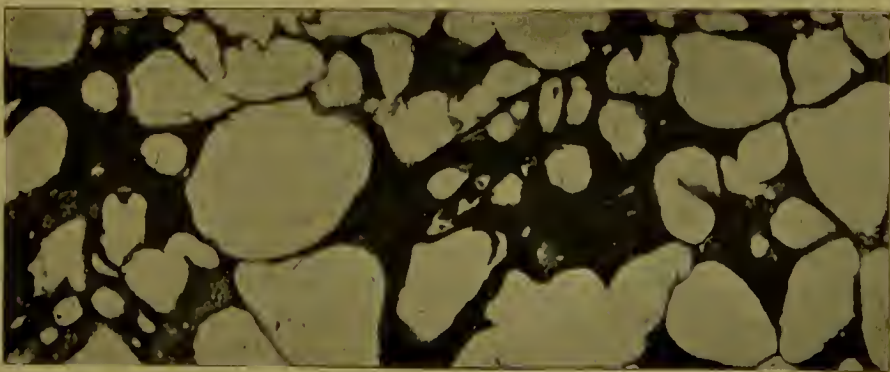
Where complications arise, the treatment is that of the complicating disease.

In cases which are protracted, a change of air, either to the country or to the sea-shore at suitable seasons, is often followed by an apparent shortening of the duration of the attack.

PROPHYLAXIS.—Pertussis is so highly contagious a disease, and may be so serious an affection in certain children, that a rigid prophylaxis should be enforced. It is the duty of those who take care of children with pertussis to see that they are isolated during the whole course of the disease.

I have here a specimen (Fig. 142), made by Northrup, of a lung taken from an infant (Case 477) under one year of age, who died during a violent attack of pertussis.

CASE 477. FIG. 142



Emphysema following pertussis. Distended alveoli often coalescing.

The section shows extensive vesicular emphysema, with great distention of the walls of the alveoli.

I have here a little girl (Case 478), four years old, who is in the fifth week of an attack of pertussis.

The intervals between the paroxysms are usually one or two hours. She has just begun to cough, and you will have an opportunity of seeing her in one of the paroxysms.



You see that after coughing a number of times she has become decidedly cyanotic, and that she is aiding the expiratory effort by bending forward and placing her hands on her knees. After the whoop has occurred and the tenacious mucus has been expelled, she obtains relief.

CASE 478.



Pertussis during paroxysm. Female, 4 years old.

The position of the child is very characteristic, as is also the swollen and congested condition of her face. When this picture is once seen, and you have heard the characteristic whoop, you will have no difficulty in making the diagnosis of pertussis.

In some cases, even in older children and where no complications are present, the attack of pertussis may be so severe as to prove serious. I saw a case of this kind in consultation with Dr. Howe, of Cohasset.

A boy (Case 479), six years old, had had pertussis for five weeks. For two weeks previous to my seeing him the cough had been so frequent and so constantly accompanied by vomiting that the child had been unable to retain any food. He was very much emaciated, and was so weak that he could not stand. This condition lasted for a week or ten days: he then began to improve, and finally recovered entirely.

**ASTHMA.**—Asthma is an affection of the lungs characterized by spasmodic attacks of dyspnoea. The disease is rare in infancy, but is not uncommon in childhood.

**ETIOLOGY.**—The cause of asthma has not been satisfactorily determined. There is a strong neurotic element in the disease, and in many cases this element is apparently hereditary. In individuals who have a tendency to the disease it may be incited by various causes, such as sudden atmospheric changes or the inhalation of irritants.

**PATHOLOGY.**—There are no known pathological lesions which characterize the disease. In cases of long duration the lesions of chronic bronchitis are often found.

**SYMPTOMS.**—The symptoms of bronchial asthma are the same in the child as in the adult. The onset is usually sudden, and generally occurs at night. A catarrhal condition of the respiratory tract, especially of the bronchi, commonly precedes the attack for some days. The child is seized with distressing dyspnoea, mainly expiratory, the respiration being accompanied by a wheezing sound. The face is anxious, and if the attack continues for some time it becomes slightly cyanotic. The respirations are not especially increased in frequency. The pulse is rapid, and when the dyspnoea is very intense it is weak. The temperature is not raised by the asthma, and where the paroxysm is prolonged it may become subnormal. The physical signs are mostly diffuse, sibilant, and sonorous râles. The attack may last for a number of hours, or even for days. The paroxysms vary in their severity, and, as a rule, are followed by considerable exhaustion. The frequency of the attacks varies; they may occur often or only at intervals of months.

**PROGNOSIS.**—The prognosis of asthma with regard to the especial attack is good. Where the disease is not hereditary the children very commonly recover from it as they approach the age of puberty. In many cases the attacks seem to depend upon some local affection of the air-passages, and the cure of these local lesions will often be followed by recovery from the attacks of asthma.

**TREATMENT.**—In the treatment of asthma, the nose and throat should be carefully examined for local diseases, as the attacks may be caused by the different forms of rhinitis, adenoid growths, or enlarged tonsils. The children should be protected from unfavorable atmospheric influences, a high, dry, inland air usually being better suited to them than sea air. In some cases, especially of a mild form, the fumes of nitre paper will give considerable relief. In very severe attacks hydrate of chloral may be given, either by the mouth or by enemata. Antispasmodics, such as belladonna and lobelia, can also be used. There is no one drug which will relieve the paroxysms of asthma except morphine, which should be used with great caution. Iodide of potassium in gradually increasing doses is in some cases beneficial. Especial attention should be paid to the general hygiene and to the diet of the child.

**PERIODIC CATARRH** (Autumnal Catarrh; Hay Fever; Rose Cold).—Closely allied to asthma is an affection of the respiratory tract occurring periodically and characterized by great irritation of the mucous membrane of the eyes, nose, throat, and bronchi. The same causes that have been supposed to produce asthma seem to be of etiological importance in periodic catarrh. These attacks usually occur in the summer months, but are generally most severe in August and September.

The onset of the attack, in contradistinction to the paroxysms of asthma,



is generally at some definite time of the year. The especial attack lasts for five or six weeks, or even longer. It is characterized by a severe acute catarrhal inflammation of the nose, eyes, throat, and bronchi. The coryza and lachrymation are in many cases excessive. As the disease progresses, the cough becomes very distressing, and the respirations are so impeded by the congested and swollen mucous membranes that sleep is interfered with, and the child's general nutrition is soon affected. There is no general remedy which controls the disease, and benefit usually can be obtained only by removing the child to a locality which is free from the causes that produce the disease.

The prognosis in children is good. The local treatment of the upper air-passages is the most likely means of obtaining a cure. If it is left untreated the disease occurs every year, so that just before the yearly attack begins it is well to have the children taken to the especial locality where it has been found that they do not suffer from the disease. In this way the impairment of their general health will be prevented, and it is possible that they will eventually cease to be affected by the disease.

Where the child cannot be removed from an irritating locality, temporary relief can be obtained from sprays of cocaine. As recommended by Wyman, the windows of the sleeping-room should be closed early in the evening and kept closed during the night. In this way the dust in the air is allowed to settle, and there is less danger that the irritating material, whatever it may be, will produce its effect when the child is asleep. As a rule, it is advisable to give the child quinine in tonic doses, beginning just before the date of the onset of the disease and continuing with it until the attack has almost run its course.

## LECTURE L.

## DISEASES OF THE PLEURA.

**PLEURISY.**—Inflammation of the pleura may be acute or chronic, and may be accompanied by an effusion, which may be serous, sero-purulent, or purulent.

Acute pleuritis, either with a simple exudation of fibrin or accompanied by fluid, is quite frequent in children. The effusion has a greater tendency to be purulent in children than in adults. It seems to follow exposure of various kinds and to be produced by a number of organisms. As a secondary affection it occurs especially after lobar pneumonia and pulmonary tuberculosis, also in the course of the acute exanthemata and in such diseases as rheumatism.

In regard to the micro-organisms which are supposed to produce pleuritis there is an evident difference in the intensity of the inflammation which follows their invasion. In the serous exudations the pneumococcus has been found most frequently, and seems to be most commonly present in the benign forms of the disease. Next to the pneumococcus the staphylococcus has been found to be present in the least virulent forms. The bacterium which has been found in the pleuritic effusions of the severest cases is the streptococcus. In those effusions which arise from tuberculosis the bacillus tuberculosis has been found.

**PATHOLOGY.**—Pleuritis is usually a unilateral disease, but may in rare cases be bilateral. The pathological conditions found in the pleurisy of children do not differ from those which occur in later life. Although localized areas characterized by the production of fibrin (dry pleurisy) are quite frequently found at the post-mortem examination, the diagnosis of this form of disease in infants and in young children is not often made during life. Where, however, large areas of the lung are involved in broncho-pneumonia, dry pleurisy quite frequently occurs, and small circumscribed areas are commonly met with in connection with lobar pneumonia. In the common form of pleurisy, where there is a production of fibrin and serum (pleurisy with effusion), a greater part of the pleura of one side of the chest is usually involved. According to Delafield and Prudden, while the inflammation is in progress the surface of the affected pleura is coated with fibrin, bands of fibrin stretch between the parietal and pulmonary layers of pleura, and in the pleural cavity there is serum in variable quantities. This serum is sometimes clear, sometimes is turbid from the presence of pus-cells and flocculi of fibrin. Both these forms of pleurisy, although differing in their clinical history, are anatomically essentially the same. In both we find, first, the exudation of fibrin and a few pus-cells either with or



without serum ; second, a gradual absorption of the serum and fibrin ; finally, a formation of new permanent connective tissue in the form of adhesions or of thickening of the pleura. Through the whole process the tissue of the pleura is but little changed. The products of inflammation, although they originate in the tissues, do not infiltrate it, but make their way to its surface, accumulate there, and undergo different changes. Variations from the regular course of the inflammation are caused by the excessive formation of the fibrin, the serum, or the pus, and by the manner in which these inflammatory products are absorbed. It is still, however, undetermined whether acute pleuritis with a serous effusion is an entirely separate disease in children from a pleuritis with purulent effusion, or whether the difference between the two diseases is merely one of degree in the amount of pus-cells present. Clinically there is a certain amount of evidence in favor of the former supposition, as an acute pleuritis with serous effusion in young children usually runs a definite benign course and is reabsorbed without becoming purulent. Empyema in young children, on the other hand, is frequently, so far as can be determined, a purulent exudation from the beginning. It is therefore better in describing pleuritis in infancy and early childhood to speak of the serous effusion and the purulent effusion as two separate diseases.

**SYMPTOMS.**—The onset of acute pleuritis with serous effusion is in many cases violent, and attended by a high temperature, increased respirations, quickened pulse, restlessness, and even pain, which in young children is usually referred to the abdomen. In infants and in young children convulsions are quite common, while in older children the symptoms are more like those which occur in adults. There is a short, painful cough, with loss of appetite, and frequently vomiting and diarrhœa. These early symptoms are usually followed in two or three days by an exudation and by a decided lessening of the pain and dyspnœa. At the same time the temperature begins to have a decided morning remission. When the exudation is large, the children lie more comfortably on the affected side, and when they are nursing they nurse most easily from the right breast if the left pleura is affected, and from the left breast if the right pleura is affected. After the serous effusion has remained for a number of days it ordinarily begins to lessen in amount, absorption takes place, and by the end of a week or ten days it becomes entirely absorbed and the child recovers. In other cases it becomes chronic unless its absorption is furthered by aspiration.

The physical signs of pleurisy before the effusion has taken place are in infants and in young children quite difficult to detect. The friction-rub is often absent and the pain is difficult to locate. It is frequent, however, to find that there is tenderness on the affected side on palpation and percussion, as the child cries more when the affected side of the chest is compressed.

When the effusion has taken place, the chief physical signs are dulness on percussion, bronchial respiration, and, if the effusion is in considerable amount, displacement of the heart. The other physical signs, such as de-

creased vocal resonance and fremitus, which are commonly met with in the plenrity of adults, are not, as a rule, sufficiently marked in infancy and early childhood to be of much value for diagnosis. Great difficulty may arise in auscultation from the finer sounds being obscured by the child's crying, but in the intervals when the child takes a breath and its cry must necessarily cease for a moment, valuable information can be obtained by the quick use of the stethoscope.

Where the effusion is sufficiently large to displace other organs, such as the liver and the spleen, the presence of the effusion is so evident from the usual signs that these displacements are not of especial value except so far as they show that the effusion is in large amount. After these large effusions have lasted for some time, and especially when they are purulent, I have met with decided bulging of the affected side.

DIAGNOSIS.—The diagnosis of pleurisy with serous effusion is to be made from lobar pneumonia and from empyema. The physical signs which in the adult are most useful in differentiating pneumonia from a pleuritic effusion are often misleading or absent in the young child. Thus, absolute dulness may occur in other conditions as well as in a pleuritic effusion, while bronchial respiration, such as is heard over a consolidated lung, may also be heard over a large effusion. The vocal fremitus may be absent in a pneumonic consolidation, and sometimes, though rarely, well marked over an effusion. Moist râles have been heard in children over an effusion, and fluid has been aspirated at a point where a friction-rub was heard. It is well known, also, that aspiration is not a conclusive means of diagnosis, for punctures have often been made where an effusion was present and yet no fluid was obtained. The change in the level of the effusion on change in position is of some value in diagnosing a pleuritic effusion from pneumonia, but is often difficult, and at times impossible, to determine in young children. The most reliable means of diagnosis in infancy and in early childhood is percussion. The area of dulness which occurs in lobar pneumonia is quite different from that which is found in cases of effusion uncomplicated by previous adhesions. If adhesions are present, these typical changes are so interfered with that the percussion becomes as unreliable a sign as the others which I have just mentioned. The younger the individual, however, the less likely are extensive adhesions to be present, and the more valuable, therefore, is the evidence of an effusion given by percussion. As has been shown by Whitney, when the effusion is small there is absolute dulness (flatness) at the base of the thorax. A friction-sound may be heard over the dull area, and respiration may be quite distinct, and sometimes accompanied by râles. Under these circumstances the diagnosis of the condition as one of effusion must depend upon the outline of the area of dulness. In determining these small areas of dulness the lower border of the two sides of the thorax must first be carefully compared by percussion, bearing in mind that the lower border of the pulmonary resonance in early life corresponds to the position of the ninth dorsal



vertebra on the right side and to that of the tenth dorsal vertebra on the left, as I have already explained to you in my lecture on development (page 122).

You must always remember that the percussion of an infant's or a young child's chest should be very light, as heavy percussion, owing to the delicacy of the thoracic walls in early life, is unreliable.

Palpatory percussion, for the same reason, gives more information when the child is crying than can usually be obtained by the sound, but, as I have said in describing the auscultation in these cases, quick percussion in the intervals of respiration is also a valuable aid to diagnosis.

Extended observations have been made on the line of percussion-dulness found in medium effusions by Ellis and Garland, and lately in small effusions by Whitney, of Denver. These investigators have shown that as an effusion increases in quantity its upper border undergoes a gradual series of changes, provided there are no adhesions.

Where the effusion is small in amount it can usually first be detected in the back. In these small effusions the upper border of absolute dulness begins at the vertebral column, extends outward horizontally for a distance which varies according to the size of the effusion, and drops in the neighborhood of the posterior axillary line by a curve more or less abrupt to the base of the thorax. As the effusion increases in size the line of dulness drops more anteriorly. Where the effusion is moderate, as where the lower half of the pleura is filled, in some cases, but not usually, a slight displacement of the heart may be noticed, and the percussion over the slightly compressed lung may give tympanitic resonance. The upper border of the area of dulness in these medium effusions is found to extend at first outward and then upward over the angle of the scapula, reaching its highest point in the axillary region. The line may then drop abruptly from the upper axilla to the base of the thorax near the apex of the heart. This line, which has been called the "letter S" curve, is characterized by having its highest point in the axillary line. When the quantity of fluid is still larger and exceeds a certain amount, the "letter S" curve is obliterated, and the resonance over the compressed lung becomes less marked. The displacement of the heart in this latter case is a most valuable sign of effusion in young children, and with careful, light percussion the gradual increase and decrease of the effusion where it is of any great extent can be determined by cardiac percussion.

You must always bear in mind the physiological dulness of the heart which I have already described as occurring in early childhood (page 123) under the lower third of the sternum. This dulness, however, is relative, and becomes much more marked and absolute where it is caused by a displaced heart.

The differential diagnosis from lobar pneumonia is greatly aided by understanding these areas of percussion-dulness which I have just described. Thus, where the diagnosis is to be made between pneumonia of a lower lobe

and a small or medium pleuritic effusion, where an effusion is present the dulness will be in the lower part of the thorax, with normal or tympanitic resonance above it; where pneumonia is present the area of dulness will often correspond to the boundaries of the lower lobe only. In like manner the area of dulness of the effusion will differ in the axillary regions and in the front of the thorax from the areas of dulness produced by the consolidation of the different lobes of the lung.

The diagnosis from empyema is very difficult, as the younger the individual the more likely are the effusion to be purulent and the early symptoms to be similar to those of the serous form of exudation. After the first week or ten days of the disease, however, where the effusion is purulent, the usual signs of absorption which so commonly occur in a serous effusion are ordinarily not found, and aspiration of the pleural cavity will then determine which form of the disease is present.

Where no bacteria are found in the fluid, where there is no history of a preceding acute pneumonia or a neoplasm of any kind, or where there is little tendency to absorption of the exudate, and where the exudate is found to contain blood, the failure to find bacteria in the exudate may be regarded as pointing strongly towards a tubercular origin.

PROGNOSIS.—The prognosis of a serous effusion, as a rule, is very favorable in infants and in young children unless one of the more virulent forms of the pyogenic cocci is present, or unless the disease is secondary to tuberculosis elsewhere and is caused by the bacillus tuberculosis. If the serous effusion tends to become purulent, the prognosis is not so good, but still, provided appropriate treatment is carried out, it is favorable. If, as in rare cases, the pleuritic effusion occurs on both sides, the prognosis becomes grave. The possibility of the presence of tubercle should be considered in these latter cases.

I have had in my service at the City Hospital a boy (Case 480), thirteen years old, who was attacked with pleuritis and a serous effusion of the left side with displacement of the heart to the right. After one aspiration the fluid was quickly absorbed, but three weeks later he was attacked with pleurisy on the right side, followed by an effusion and displacement of the heart to the left. This effusion was absorbed without aspiration, and the boy was discharged from the hospital well and strong, with both lungs apparently in a normal condition.

Where the effusion is very large and the heart is much displaced, there is always the danger of a fatal issue from asphyxia, and the prognosis depends upon whether the effusion can be controlled by aspiration and the heart thus be kept in normal position. A case which illustrates the danger of these large effusions accompanied by displacement of the heart came under my care at the Children's Hospital:

A boy (Case 481), four or five years old, entered the hospital with a large effusion in the left chest. The heart was displaced to the right, and upward as far as the second interspace to the right of the sternum. He was cyanotic and gasping. On aspirating the



chest and removing a large quantity of fluid, the heart reassumed its normal position under the sternum. On the following night the boy died suddenly, the effusion having rapidly accumulated and having again displaced the heart.

Cases of this kind should warn us that a pleuritic effusion of any extent in a young child should be watched continuously, and that aspiration should be performed where there is indication of an increase in the intrathoracic pressure.

Where the pleurisy is secondary to other diseases, such as rheumatism and scarlet fever, the prognosis is not so favorable: the effusion is not apt to be absorbed so readily, and is more likely to become purulent. The prognosis is also rendered more unfavorable in these cases by the prolonged pressure upon the lung, with its corresponding ill effects upon the general condition of the child. The dangers which arise from the development of tuberculosis must also be borne in mind.

TREATMENT.—The treatment of pleuritis during the early days of the attack, before an effusion of any considerable extent has appeared, should be directed to the relief of the pain by a flannel bandage closely applied to the thorax, so as to allow the ribs to move as little as possible in respiration. Sometimes an occasional dose of *tinctura opii camphorata* will also be needed to make the child comfortable. After the effusion has increased, the child should, if possible, be kept in bed. There are some cases, however, where a child with a considerable effusion in its pleura will feel well and bright, and will play about its nursery without showing any especial symptoms of discomfort. I have met with instances of this kind where, excepting that it was pale and had a poor appetite, the child seemed bright and active, and yet it had a pleuritic effusion large enough to displace the heart.

In mild cases, after the effusion has attained its maximum, you should carefully examine the child each day, to see whether there is a rapid increase in the fluid, which by displacing the heart and causing dyspnoea would render necessary immediate relief, or whether the fluid has begun to be absorbed. In the latter case an expectant treatment is all that is required. In the former case, or if absorption of the fluid is delayed for two or three weeks, the chest should be aspirated. A bacteriological examination of the fluid removed should then be made, to determine which form of organism is present in the exudate. If one of the more benign forms of bacteria is present, such as the pneumococcus, or if the fluid is found to be serous, no further treatment will be required, unless there be a reaccumulation of the fluid, in which case a second aspiration will be indicated. If, however, the streptococcus is found in the exudate, the case must be watched very carefully, as it is more likely to become purulent and to need radical treatment.

As the unfavorable symptoms in a pleuritic effusion arise mostly from intra-thoracic pressure, relief from the pressure by aspiration is indicated rather than by the use of drugs, which cannot be depended upon.

The point of aspiration should usually be in the fourth or fifth interspace in the axillary line, or a little farther back.

**Purulent Pleuritis (Empyema).**—Empyema is a purulent effusion into the pleural cavity. In the first three or four years of life it is much more common than a serous effusion.

The cause of these purulent effusions is the same, so far as we know, as that of serous effusions. The same organisms are present in the two forms, and they are also frequently present when purulent pleurisy is secondary to a number of diseases, the most prominent of which is lobar pneumonia.

Usually the whole pleura is involved, encysted empyemas in infants and young children being rare.

The disease when primary may be acute in its onset, and may simulate closely the initial stage of lobar pneumonia. In other instances it is slow and somewhat insidious in its development. The pulse and respirations may be quickened, but after the early days of the disease they are often very little raised. There is nothing characteristic in the temperature of an empyema, and the diagnosis usually can be made only from the knowledge that the younger the individual the more likely is pus to be present. This, however, can be determined definitively only by means of the aspirator.

The physical signs are the same as in a serous effusion. The absorption of a purulent exudate without surgical interference is very rare. I have occasionally met with cases where one aspiration was all that was necessary, and where, seemingly, complete absorption took place.

Where cases of empyema are left untreated, a spontaneous opening usually takes place through some portion of the thoracic walls, but the exudate may also find its exit through the lungs by opening into one of the bronchi or perforating in other directions. I have met with cases where the diaphragm was perforated and the point of exit of the pus was in the region of the umbilicus. Where perforation does not occur, the pus is partially absorbed, adhesions are formed, and sometimes great deformity of the chest follows, which may result in a marked degree of lateral curvature of the spine as well as in great contraction of the chest.

After the first aspiration, unless absorption occurs within a week, and especially if one of the more virulent forms of bacteria is found in the exudate, a radical operation is the best method of treatment. Where this is performed early in the disease, the prognosis is very good in infancy and early childhood, unless the empyema is of a virulent form or is secondary to some incurable disease, such as is caused by the bacillus tuberculosis. The pleural cavity should be thoroughly drained by means of drainage-tubes. In many cases, especially in children over two or three years of age, resection of one or two ribs gives the best results. Although in some cases a rapid cure in two or three weeks follows the operation, yet the recovery is often prolonged for many months, even where strict antiseptic precautions have been taken at the time of the operation.



As the treatment of empyema is essentially surgical, I shall not enter into its details.

This little girl (Case 482), eleven years old, was attacked six weeks ago with a chill followed by vomiting. She then had a short, dry cough. For the past few days she has complained of pain in the lower part of the right chest. She has been feverish, has lost in weight and in appetite, and her respirations have been painful. She lies most comfortably on her back and on her left side. A pleuritic friction-rub has been heard in the right axillary region. Her lips and cheeks are slightly cyanotic.

CASE 482.



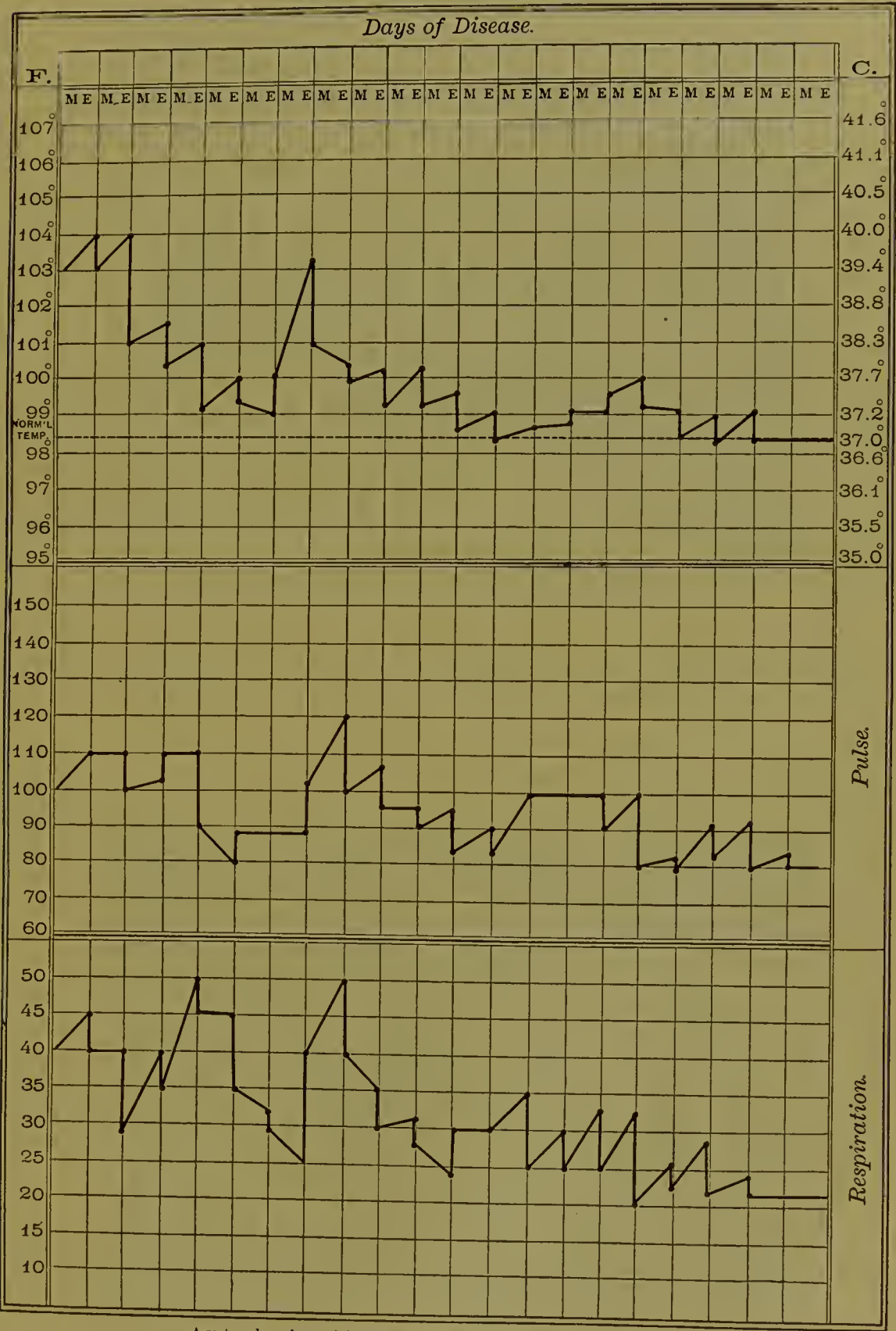
Acute pleurisy with serous effusion. Female, 11 years old. The line of the upper border of the effusion, the area of cardiac dullness, and the margins of the ribs are marked in black.

Her tongue is somewhat coated. The *alæ nasi* are working, and, as you see, the dyspnoea is so marked that she has to sit almost upright in bed. The percussion and auscultation of the left lung show nothing abnormal. The resonance is fair over the upper part of the right front and back. There is absolute dullness from about the fifth dorsal vertebra in the right back to the base of the lung. This dullness extends into the axillary region, where it reaches its highest point, and then gradually descends to the right parasternal line on a level with the fourth costal cartilage. Over this area of dullness respiration is markedly diminished. No friction-rub is heard. The vocal and the tactile fremitus are diminished. The impulse of the heart is found in the fourth interspace, 1 cm. ( $\frac{3}{8}$  inch) to the left of the mammary line. The heart-sounds are normal. There does not appear to be any displacement of the liver. An examination of the urine shows it to be acid, to have a specific gravity of 1022, to be of normal color, and to contain no albumin. The chlorides are normal. The physical signs are those of a pleuritic effusion of the right side with displacement of the heart to the left.

(Subsequent history.) During the following week the area of absolute dullness gradually decreased, and an exploratory aspiration showed the fluid to be serous. Three weeks

from the time when she entered the hospital, and nine weeks from the beginning of the attack, the dulness on percussion gradually disappeared, auscultation showed the respiration

CHART 42. (CASE 482.)



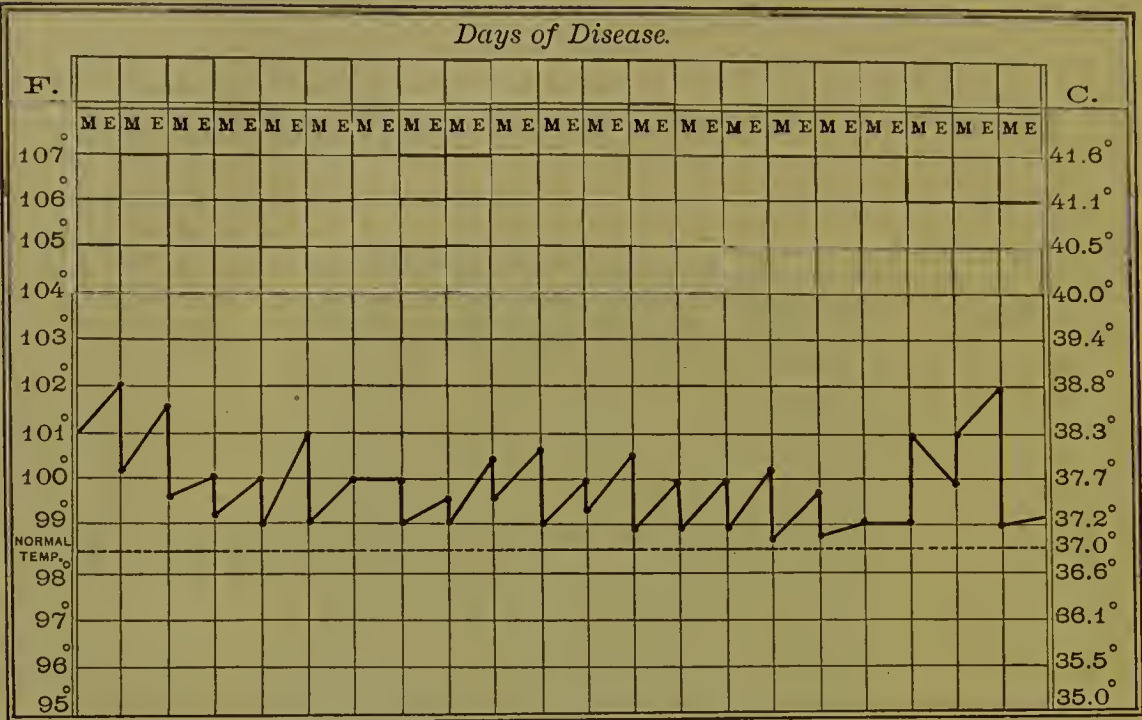
Acute pleurisy with serous effusion. Female, 11 years old.

to be normal, and the heart resumed its normal position. This chart (Chart 42) shows the temperature, pulse, and respiration while she was in the hospital.



This chart (Chart 43) shows the temperature for twenty-one days in a case (Case 483) of serous effusion in the pleura, where in the beginning 165 c.c. (5½ ounces) of fluid were withdrawn from the chest.

CHART 43. (CASE 483.)



Acute pleurisy with serous effusion. Male, 4 years old.

The fluid reaccumulated, so that absolute dulness was found over the whole right side of the chest in front and behind, but aspiration did not have to be resorted to again, and complete absorption took place thirty days from the beginning of the attack.

A number of cases have been reported in which a purulent effusion has been treated by aspiration and has seemingly disappeared entirely without a radical operation. These cases should be borne in mind when treating purulent pleuritic effusions. An infant (Case 484), seven weeks old, with empyema, at the Boston City Hospital, in the service of Dr. Doe, recovered entirely after one aspiration.

The initial stage of empyema often closely simulates pneumonia.

A case illustrating this fact has come under my observation, where a boy (Case 485), three years old, and previously well, was attacked with pain in the left side, with a chill and with dyspnoea. A physical examination made on the third day of the attack showed the right lung to be normal. On the left side of the chest there were absolute dulness, diminished respiration, and increased vocal resonance, and fine râles were heard from the fifth rib to the base of the lung, both in front and behind. Two days later an exploratory aspiration showed that the physical signs were caused by an empyema.

Another case which illustrates the difficulty in diagnosing a purulent effusion in the pleura in the early days of the disease is the following :

A girl (Case 486), four years old, was suddenly attacked with cough, and pain in the right side. The temperature was 40.5° C. (105° F.). The respirations were quickened, and the pulse was rapid. Nothing abnormal was detected on physical examination. On the following day the general symptoms disappeared, and the temperature fell to 38.8° C. (102° F.). In another day the temperature fell to 37° C. (98.6° F.), and the child seemed

CASE 488.

I.



Purulent pleuritis of right side. Female, 1½ years old. The black line shows the upper border of the effusion.

II.



Scar left after operation for purulent pleuritis.





bright and well. On the following day, however, the temperature rose to 40° C. (104° F.), absolute dulness was detected in the right axillary region, and an exploratory aspiration showed the presence of pus.

Here is a little girl (Case 487), three years old, who two years ago had an attack of some pulmonary disease accompanied by fever. Since then she has been delicate and has coughed a great deal. Her cough has increased in the last few weeks, but she has not lost in weight nor had any other abnormal symptoms. She is pale, and the cervical, axillary, and inguinal glands are enlarged. Her fingers are markedly clubbed. She shows a peculiar lateral curvature of the spine, which cannot be made to disappear by traction. The right side of the thorax expands normally, the left side scarcely at all. There are hyperresonance over the right lung, no râles, and compensatory respiration. The left lung is apparently atelectatic, and shows dulness everywhere except in a small triangular area at the inferior angle of the scapula. This deformity of the thorax is probably the result of an empyema which occurred two years ago and was not properly treated.

Here is an infant (Case 488), one and a half years old, who entered the hospital with a history of an acute attack, characterized by fever, cough, and dyspnoea. Physical examination showed nothing abnormal on the left side of the chest, but on the right side there was absolute dulness, with bronchial respiration. No râles were heard anywhere in the lung.

The upper border of the area of dulness I have marked with a black curved line, and you see how, beginning at about the fourth dorsal vertebra, it gradually rises as it approaches the axillary line and then falls as it approaches the right parasternal line. The child's

## CASE 489.



Recovery from empyema. Male, 10 years old, showing scar eight years after operation.

right arm has been turned upward and forward, in order that the ribs shall be sufficiently separated for the introduction of the trocar preparatory to an operation for the radical cure of the empyema. An exploratory aspiration has already shown the presence of a purulent effusion in the pleura.

(Subsequent history.) The child was operated upon by Dr. Burrell, an incision being made in the mid-axillary line on the right side, and about 1.4 cm. ( $\frac{1}{2}$  inch) of the seventh



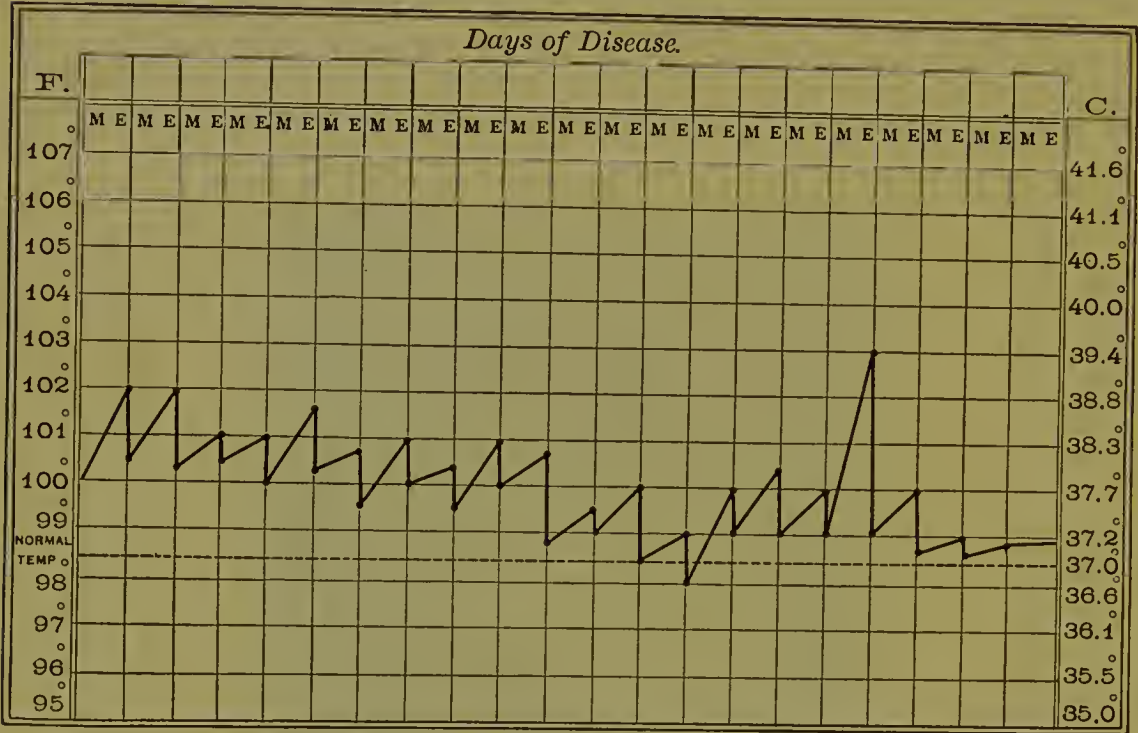
rib was resected. Nearly two quarts of pus were evacuated, and the pleural cavity was washed out with a boracic acid solution, a drainage-tube inserted, and a baked dressing applied.

This picture (Case 488, II.), taken some months later, shows the scar which was left after the operation.

This boy (Case 489, page 1017), ten years old, had a purulent effusion on the right side when he was two years old.

On entering the hospital with a history of having been sick for a number of weeks, there was found to be absolute dulness over the whole right side of the chest, with displacement of the heart to the left. Aspiration showed the dulness to be produced by a purulent effusion in the pleura. I made a permanent opening, and after thirty-six days the boy recovered, and was discharged from the hospital with the lung apparently normal. I show him to you to-day in order that you may see how the scar looks after a number of years. You see that the right side of the chest is equally expanded with the left, and that no deformity has resulted from an extensive empyema.

CHART 44. (CASE 489.)



Purulent pleuritis. Male, 2 years old.

This chart (Chart 44) shows the temperature during the twenty-one days previous to the removal of the drainage-tube.

## DIVISION XVII.

### DISEASES OF THE HEART AND PERICARDIUM.

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#### LECTURE LI.

##### DISEASES OF THE HEART.

CARDIAC disease in infancy and early childhood may be divided into congenital or acquired, developmental or inflammatory, functional or organic, acute or chronic. In this early period of life cardiac disease has certain characteristics in which it differs essentially from those which are met with in later life. One of these characteristics is that there is a more decided tendency to recovery than at a later period. Another is that, owing to the undeveloped condition of the infant and young child, interference with the growth of other organs and parts of the body may more easily result from diseases connected with the circulation than is possible in the case of the fully-developed adult. Thus, there are certain anatomical facts connected with the ossification of the sternum which become of great importance in connection with cardiac disease. Deformities of the thorax may result from the continued pressure of the enlarged heart on the soft and pliant sternum and costal cartilages of the young subject. These deformities do not arise merely where the individual is rhachitic, but may also depend upon the stage of development at which the cardiac disease begins. The deformity is more or less pronounced in inverse ratio to the age and in direct ratio to the time during which the cardiac disease has existed. The shape and extent of the deformity are also dependent on the degree of ossification which has taken place in the sternum. In young infants, where the entire sternum, as I have described in a previous lecture (page 71), is in a cartilaginous condition, the intra-thoracic pressure from an enlarged heart may cause a bulging of the whole front of the thorax. This may occur during the first year, and even up to the third year. As the child grows older, the manubrium and the second piece of the sternum become ossified and offer more resistance, while the third piece of the sternum, still remaining in a semi-cartilaginous condition, may be tilted. This may occur in children in whom the cardiac disease has not developed until the fourth, fifth, or sixth year. I have had under my care a child seven years old who at the age of five years



had articular rheumatism with resulting cardiac hypertrophy, and who presented this displacement of the third piece of the sternum. No other signs of rhachitis were detected. The middle period of childhood is also a peculiarly unfortunate one for the occurrence of cardiac disease, because the heart grows so rapidly at this period that it requires a proportionately greater amount of intra-thoracic space for the normal performance of its function than it does later.

In addition to the injury which may be done to the thoracic walls by an enlarged heart, we must consider the interference with the normal uniform expansion so necessary for the growing pulmonary tissue, and the consequent loss of the elasticity which plays so prominent a part in the establishment of the equipoise which should exist in a perfected respiratory apparatus.

The occurrence of *diseases of the blood-vessels* is rare in infancy and early childhood in comparison with later life. *Aneurism* is rare. A *narrowing of the isthmus aortæ* is more common, and is one of the most marked of the congenital defects of the blood-vessels. Sometimes there is an *absence of the isthmus aortæ* during foetal life. The compensation for this defect takes place by an increased action of the left ventricle and the establishment of a collateral circulation between the subclavian artery and the thoracic and the abdominal aorta. These malformations exert in varying degrees an influence on the heart, as the infant grows older, from increased blood-pressure.

**CONGENITAL DISEASES OF THE HEART.**—Congenital diseases of the heart are somewhat obscure in their etiology, but usually they result either from an interference with the normal development of the organ or from endocarditis, or from a combination of both. In order to understand these congenital lesions you must remember the chief points in the mechanism of the foetal circulation, which I explained to you in a previous lecture (page 19). I then told you that the parts of the foetal circulation at birth which were of most importance in reference to diseased conditions of the heart and great blood-vessels later were the foramen ovale and the ductus arteriosus. I also told you at what period after birth they disappeared. Where these remains of the foetal circulation, which are normal during intra-uterine life and for a short period afterwards, continue as the infant grows older, they become abnormal and interfere with the equilibrium of the circulation.

Where the development of the heart has been interfered with in intra-uterine life, there results another set of malformations, the chief of which are an open ventricular septum, a transposition of the great vessels connected with the heart, and various malformations of the valves of the heart. Where, again, an inflammatory condition has taken place in intra-uterine life (foetal endocarditis), various other morbid conditions result, the most common of which are connected with the pulmonary artery, causing stenosis or atresia, a narrowing of the conus arteriosus, and various malformations of the tricuspid valve and other orifices of the heart.

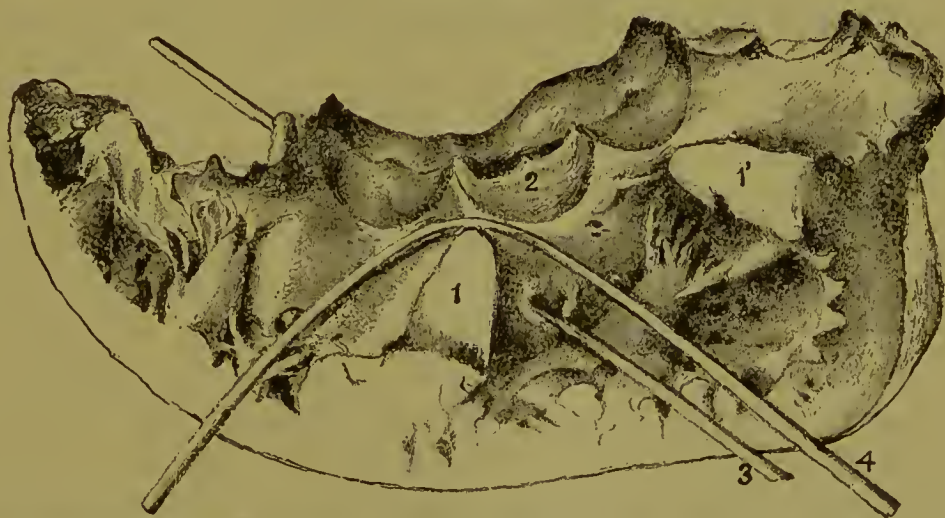
The form of inflammation of the endocardium which occurs in intra-uterine life is the chronic or sclerotic variety. Verrucose endocarditis is rare. (Osler.)

A deficient filling of the left side of the heart in early life, such as occurs in cases of atelectasis, foetal pneumonia, or foetal endocarditis, especially where stenosis of the pulmonary artery has resulted, may delay the closure of the foramen ovale and of the ductus arteriosus, which under these circumstances act as safety-valves. This is true also of the delay in the closing of the intra-ventricular septum, which is often of great aid in preserving the equilibrium of the circulation. In congenital cardiac disease it is usually the right side of the heart that is affected. The most common congenital cardiac lesions are an affection of the pulmonary artery, an open foramen ovale, an open ventricular septum, and an open ductus arteriosus.

The lesions most commonly found in connection with the pulmonary artery are stenosis of the pulmonary orifice, atresia of the orifice and of the artery, and stenosis of the conus arteriosus. Stenosis of the pulmonary orifice usually results from foetal endocarditis, though it is possible that it may be the result of faulty development. The complete obliteration of the orifice of the beginning of the pulmonary artery is common, though not so frequent as stenosis of the orifice, and is probably of developmental origin.

I have here a specimen (Fig. 143) which was taken from a child (Case 490) with congenital cardiac disease under the care of Dr. Northrup. It illustrates this malformation of the pulmonary orifice.

FIG. 143.



Congenital cardiac disease. Male, 4½ years old. Right and left ventricles laid open by two cuts. Stenosis of pulmonary orifice. Incomplete septum ventriculorum. 1 and 1', septum ventriculorum cut across; 2, aortic valves; 3, probe passing through narrowed pulmonary orifice; 4, bent probe passing through right ventricle to left through opening in septum ventriculorum.

The specimen was taken from a boy four and a half years old, who during life had shown cyanosis, clubbed fingers, and at times severe dyspnoea. The physical signs in connection with the heart were a fine wave perceptible to the eye at the left third interspace, a soft, purring thrill over the base of the heart, cardiac pulsation 1.4 cm. (½ inch) outside of the left mammary line, and cardiac dulness from the right sternal margin to the left mammary line,

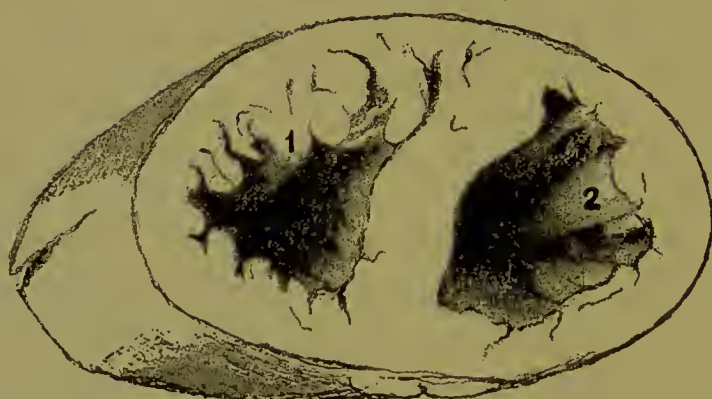


with no dulness to the right of the sternum. A loud, harsh systolic murmur was heard over the left margin of the sternum, most marked at the second left interspace and third rib, and not transmitted to the left or along the aorta.

The pulmonary artery was abnormally small, the aorta was abnormally large, the conus arteriosus was practically obliterated at the pulmonary orifice, and the ventricular side formed a ring of white cicatricial tissue  $\frac{1}{2}$  em. ( $\frac{1}{8}$  inch) in diameter.

Here is a specimen (Fig. 144) of the same heart with the apex cut away so as to show the relative thickness of the ventricular walls and the greatly thickened septum ventriculorum.

FIG. 144. (CASE 490.)



Transverse section of heart near apex.—1, right ventricle; 2, left ventricle.

The right ventricle is markedly hypertrophied. The left ventricle is normal. The ventricular septum is greatly hypertrophied. In this case the ductus arteriosus was impervious and the foramen ovale practically closed. A foetal endocarditis had taken place before the septum ventriculorum had closed. The endocarditis caused contraction of the conus, and the blood being forced from the right ventricle through the imperfect septum prevented the latter from closing. This provided a safety-valve, which, as usually happens in this form of malformation, allowed the child to live longer than is common in other congenital cardiac malformations. The aorta, receiving a direct stream from both ventricles, was distended; the pulmonary artery, receiving but little, remained small. It is interesting to note in this case that the child passed through an attack of pertussis and measles without serious results. It died ultimately of abscess of the brain.

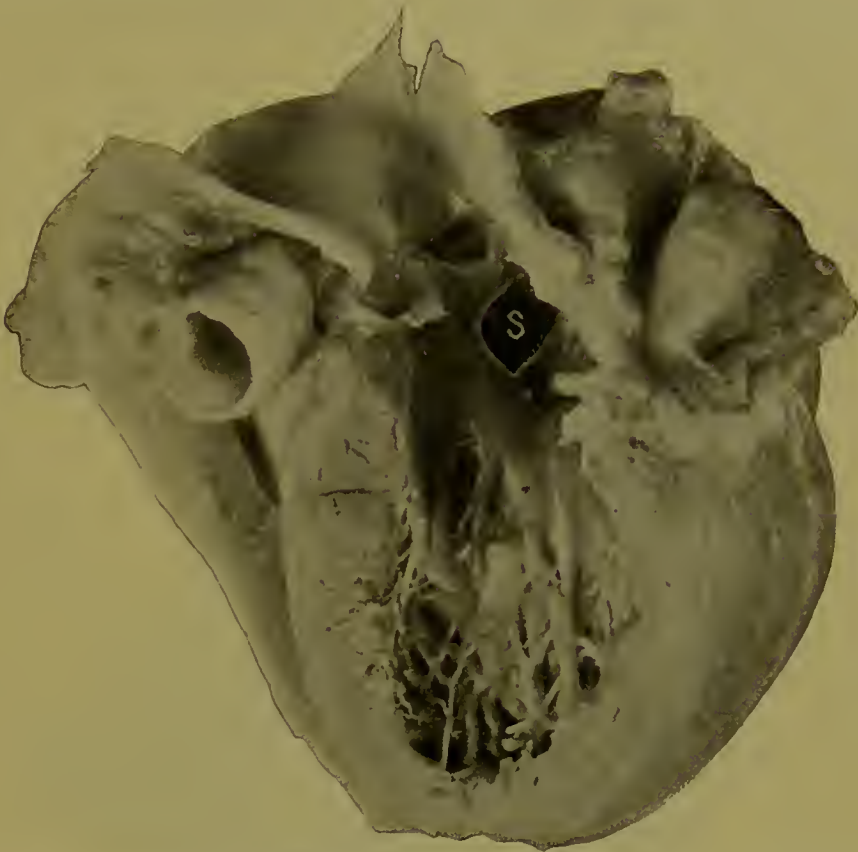
There may also be dilatation of the pulmonary artery, as in a case reported by King, where the pulmonary veins united to form a trunk of the same size as the artery and emptied into the right auricle. In this case cyanosis and oedema were present at times, and there was œdema of the face, hands, and feet. There was also icterus, apparently arising from cirrhosis of the liver, which was present.

Premature closure of the foramen ovale has been met with, but is ex-

tremely rare. I have already shown you this specimen (Fig. 6, page 42) of an infant's heart with an open foramen ovale.

Here is another specimen (Fig. 145), which shows a small opening in the ventricular septum.

FIG. 145.



S, unclosed ventricular septum. Female, 10 months old. Warren Museum, Harvard University.

In this case there was also an open foramen ovale, but no other malformation. The infant, after showing the usual progressive signs of congenital cardiac disease, died suddenly. There was no history of cyanosis.

The fourth common congenital cardiac imperfection, an open ductus arteriosus, which I have just referred to, is shown in this specimen (Fig. 146, page 1024).

This heart, which has been left attached to the lung, was taken from an infant (Case 491), sixteen days old, who was apparently healthy at birth and presented no symptoms of cardiac disease.

When the infant was five days old it was noticed that it would sometimes become slightly cyanotic. At this time its temperature rose to  $39.4^{\circ}\text{C}$ . ( $103^{\circ}\text{F}$ .). A physical examination showed nothing abnormal, and nothing abnormal was seen on inspection. The area of cardiac dulness was normal, and no murmurs were detected. A day or two later the temperature became normal; the cyanosis increased somewhat, but was intermittent and of a very slight degree. At times the skin would become cool. A few days later there was slight intestinal disturbance. When sixteen days old, without any other symptoms having developed, the infant died suddenly. The post-mortem examination made by Dr. Cutler showed this widely open ductus arteriosus. The foramen ovale is also open. There are no other lesions, such as stenosis of the pulmonary artery, open ventricular septum, or lesions of the valves. The heart is of normal size. As you see, there are no signs of the obliterative endocarditis usually found at this age in the ductus arteriosus.



The ductus arteriosus, as I have explained to you in a previous lecture (page 21), should gradually be obliterated within the first two weeks of extra-uterine life. Interference with this normal involution is not very uncommon, rarely occurs alone, and is usually found in connection with lesions of the pulmonary artery or narrowing of the isthmus aortæ. Sometimes the process of obliterative endarteritis, which has been shown by Dr.

FIG. 146.



D, open ductus arteriosus. Male, 16 days old. Warren Museum, Harvard University.

J. C. Warren to be the method by which the closure of the lumen of the ductus arteriosus is accomplished, extends to the aorta and causes stenosis of the isthmus aortæ. Again, the duct, in closing and retracting, pulls the aorta and tends to narrow that vessel, thus increasing the arterial tension. During foetal life stenosis of the isthmus aortæ does not produce much disturbance in cases where the ductus arteriosus can carry the blood to the descending aorta. At birth, however, in these cases, unless the ductus arteriosus remains pervious, serious symptoms arise, and, if life be prolonged, hypertrophy of the left ventricle takes place, and the arterial blood has to be conveyed to the descending aorta by means of a collateral circulation which is established between the branches of the subclavian arteries and the branches of the thoracic and abdominal arteries. Premature closure of the ductus arteriosus during foetal life has been met with, but is a rare condition. Very rarely the ductus arteriosus may be entirely absent.

Imperfections of the tricuspid orifice are more rare. Lesions of the mitral valve are very rare in intra-uterine life. Those of the aortic orifice are rare in comparison with those of the pulmonary orifice, but are of the same nature,—that is, they may be developmental or inflammatory.

The duration of life where there are intra-uterine lesions of the aortic orifice is not nearly so long as where the pulmonary artery is affected.

Transpositions of the aorta and pulmonary artery are very commonly met with in connection with other congenital defects, such as spina bifida or hydrocephalus, but may occur in infants who are otherwise normally developed. In these cases the duration of life is almost invariably short.

Lesions of the valves vary greatly in their extent and kind.

On the boundary line between developmental and inflammatory conditions of the heart is a class of cases in which small hæmatomata are found on the valves. These hæmatomata appear just before or just after birth, and in the process of disintegration through which they pass may cause a contraction of the valvular tissue, and thus eventually produce the same symptoms that usually result from the more common valvular imperfections.

Although these various abnormal conditions may be found alone, yet they generally occur in combination with each other, and all kinds of transpositions and malformations of the vessels are at times met with.

There are various malformations of the heart which occur at an early period of foetal development, and which are of pathological rather than clinical interest. Of these I might mention cases where there are one auricle and one ventricle (*cor biloculare*) or one ventricle and two auricles (*cor triloculare*), as well as a case which has come under my notice, where the heart had a double apex, the right apex lying in the fourth interspace to the right of the sternum, and the left apex lying in the fourth interspace to the left of the sternum.

**SYMPTOMS.**—Although in some cases the symptoms of congenital cardiac disease are very indefinite, and the disease may be masked for a number of months, yet in a large number of cases they soon become evident. The typical symptoms of congenital cardiac disease are cyanosis and attacks of dyspnœa amounting at times to suffocation and atrophy. As the disease progresses, the fingers often become club-shaped, the nails blue, and the skin cool. In connection with these rational signs there is usually an evident pulsation in the cardiac region, with bulging of the præcordia. Where the obstruction caused by the lesions is sufficient to produce hypertrophy and dilatation of the heart, an increase in the area of cardiac dulness is found. Diffuse cardiac murmurs are heard often over the whole chest, but usually have their maximum intensity towards the upper part of the sternum, and are commonly systolic in time.

The most common symptom is cyanosis. Remember that cyanosis may arise from incomplete oxygenation of the blood, and not merely from the mixture of the venous and arterial currents. Where cyanosis is present to any extent there is usually some malformation of the pulmonary artery or its valves. Well-marked congenital malformations may be present with no symptoms whatever. There may be an entire absence of cyanosis; there may be no increased area of dulness and no murmurs; and I have met with instances where the infants seemed to be thriving, and showed neither labored



breathing nor physical signs of disease up to within a few hours of death, and yet where a number of cardiac malformations were found at the autopsy. Although, as a rule, the symptoms occur at a very early period of extra-uterine life, yet quite frequently they are so mild in their character that they are not noticed especially, as is the case when they appear only when the infant is much excited or is crying. Again, the cardiac symptoms may not be prominent enough to attract attention until the infant is old enough to exert itself sufficiently, as by creeping or walking, to interfere with the equilibrium of its circulation. At times another disease, especially bronchitis or pneumonia, may precipitate the cardiac symptoms. Again, it is quite common for endocarditis to develop in a heart in which a congenital malformation is present, and the diagnosis between a congenital and an acquired cardiac affection then becomes necessary, and is accompanied by many difficulties. As an illustration of how congenital cardiac disease can be masked for a number of weeks, I shall report to you a case which has lately come under my care.

This infant (Case 492) was apparently healthy at birth, and a careful physical examination showed nothing abnormal in the thorax. There was no cyanosis noticed, the skin being of a normal color. When it was sixteen days old it refused to take the breast, and in the afternoon seemed somewhat cold, was slightly cyanotic, and had a temperature of 35.2° C. (95.5° F.). An examination of the heart detected nothing abnormal. A few drops of brandy were given to it, and after several hours the skin became warm, the respirations normal, and it took its food as usual. Early in the following morning the quickened respiration returned, the temperature rose to 37.7° C. (100° F.), it refused to take its food, failed rapidly, and died in the afternoon.

The examination of the heart by Dr. Mallory showed a large open foramen ovale and an absence of the upper part of the intra-ventricular septum below the aortic valve. The beginning of the aorta for a distance of 1 cm. ( $\frac{3}{8}$  inch) was dilated into a spherical pouch, from which were given off (1) the aorta without any branches before the intercostals, thus supplying only the lower part of the body, (2) a large vessel to the right lung, and (3) a large vessel to the left lung. From the upper part of the right ventricle was given off a large vessel which divided 1.4 cm. ( $\frac{1}{2}$  inch) above the pulmonary valve into a large vessel on the right side and two smaller ones on the left. The large vessel apparently corresponded to the innominate, and the other two vessels to the subclavian and common carotid of the left side. By these vessels blood was supplied to the head and upper extremities. There was no communication between the arterial and pulmonary vessels, as the ductus arteriosus was absent. The cause of the dilatation of the beginning of the aorta was a thickening and narrowing of the vessel for 8 mm. ( $\frac{1}{4}$  inch) just beyond the dilatation. The heart was enlarged, but not especially hypertrophied.

There was a general streptococcus invasion, for which no cause could be found. The cord had come away at the usual time without leaving any abnormal condition in the neighborhood of the umbilicus.

**DIAGNOSIS.**—Although from what I have just told you concerning the symptoms it is usually possible to make a diagnosis of congenital cardiac disease, yet when we consider the variety of lesions which may occur, and the combination of different lesions which may be present, you will understand that a diagnosis of the especial lesion is, as a rule, impossible.

Bearing in mind the mechanism of the foetal circulation (Diagram 1, page 19) and the connection which an enlargement of the heart has with

especial lesions, we can sometimes arrive at an approximately correct diagnosis; but no reliance can be placed upon the locality or sound of the cardiac murmurs, as such murmurs may be produced by very trivial lesions, and may be absent where the lesions are most pronounced.

**PROGNOSIS.**—Where the lesion is connected with the pulmonary artery, and there is an open ventricular septum to act as a safety-valve, the equilibrium of the circulation may be retained to such a degree that the child will live for a number of years. Where the only malformation is an open foramen ovale, life may be prolonged for many years. Where, however, other malformations are present, especially of such a grade as to overcome the compensatory power of the heart, death generally takes place at an early period. Where there is transposition of the main arterial trunks, the infant usually lives but a short time. Infants and children with congenital disease of the heart are very apt to die suddenly.

Death ordinarily results from some affection of the lung, sometimes from hæmoptysis, and it is quite common for tuberculosis to develop in these cases of congenital cardiac disease.

In some rare cases the compensatory power of the heart is so great that the equilibrium of the circulation is maintained, and adult life may be reached.

**TREATMENT.**—The treatment of congenital disease of the heart is essentially hygienic and symptomatic. The infants should be carefully protected from atmospheric changes which would be likely to produce bronchial irritation, as in many cases bronchitis appears to play an important part in interfering with the maintenance of the equilibrium of the circulation and in destroying compensation. In a number of cases I have found that the administration of digitalis in small doses and with the greatest caution is valuable when hypertrophy has begun to fail and dilatation to increase. Where the dyspnoea is distressing, a few drops of aromatic spirit of ammonia will often give relief. Stimulants are usually indicated.

Freedom from excitement and over-exertion should be constantly enforced, but the child should be kept in the open air as much as possible.

Here is another infant (Case 493), three months old, in whom the most striking feature of its congenital cardiac disease is extreme wasting. It has a cardiac murmur at the base of the heart, and is, as you see, slightly cyanotic. You will notice that the cyanosis sometimes affects the mucous membrane of the mouth, and that the nails are blue. A harsh systolic murmur can be detected at the base of the heart. At times the infant has serious attacks of dyspnoea and suffocation, but by simply placing it on its right side immediate relief is obtained from these symptoms, this procedure evidently bringing into action a safety-valve by which some overtaxed portion of the circulatory mechanism is temporarily freed from its burden.

**ACQUIRED DISEASES OF THE HEART.**—Acquired diseases of the heart may be functional or organic, acute or chronic.

**FUNCTIONAL.**—Functional affections of the heart do not usually occur until the later years of childhood. Functional cardiac disturbances may



arise from anæmia of the nervous centres and from cardiac irritants, such as tea and coffee. They are significant symptoms in the course of such neuroses as exophthalmic goitre.

In these functional cases there are no pathological conditions beyond a weakened condition of the muscles of the heart, and possibly, at times, a slight degree of dilatation of its cavities.

The symptoms are palpitation, a weakened irregular pulse, attacks of dyspnœa and fainting, and sometimes cardiac murmurs which are seemingly hæmic in their nature.

A marked example of this class of cases was a boy (Case 494), eight years of age, who came under my care with attacks of fainting, palpitation, and dyspnœa. He was taken from school and made to play all day in the open air, and in a few weeks these symptoms disappeared entirely.

A considerable quantity of tea is given to some children at as early an age as four or five years, and this often leads to functional cardiac disturbance. A striking example of this class of cases was seen by you at one of my previous lectures (page 469, Case 201).

In these functional cases the subjective symptoms are more apt to be marked than where there are organic lesions.

ORGANIC.—Organic diseases of the heart may be of *mechanical* or of *inflammatory* origin, and may also be *primary* or *secondary*. I have arranged this table (Table 110) showing the various conditions under which organic cardiac disease may arise in early life, and shall ask you to examine it before I speak of the various diseases.

TABLE 110.

Acquired Organic Cardiac Disease.			
Mechanical.		Inflammatory.	
Dilatation.	Hypertrophy.	Endocarditis.	Myocarditis.
Primary.	Secondary.	Primary.	Secondary.
Over-exertion. Puberty.	Pericardial and pleuritic adhe- sions. Any infiltration of lung-tissue. Pertussis with its accompanying emphysema and atelectasis. Increased blood- pressure, as from renal disease or narrowing of the aorta.		Rheumatism. Chorea. Acute exanthemata (scarlet fever). Diphtheria. Pneumonia. Endocarditis recur- rens (from old cardiac malforma- tions or lesions).

Organic diseases of the heart are more apt to attack the left side of the heart than the right. I shall not dwell in detail on the various physical

signs of cardiac disease, such as murmurs, thrills, and dulness, as they are very similar to those with which you have been made familiar in your study of the adult's heart. The importance of recognizing the relative size and position of the heart at different ages I have already spoken of in my lecture on development (page 122), and I shall therefore refer you to what I said on that occasion. There are certain differences, however, between the symptoms of cardiac disease in infancy and early life and those in later life. In young children murmurs are more apt to be diffuse than in adults, often being heard over the entire chest; and the rate and rhythm of the heart are so easily disturbed by nervous influences as to be of little diagnostic value. Progressive emaciation is a symptom which is apt to appear speedily. An enlarged heart dependent on adhesions from a preceding pericarditis is more common in early life than in adults, while compensation, as I have already told you, is much more readily acquired.

I have had children with cardiac disease presented for treatment at my children's clinic one year with cardiac symptoms so severe that they had to be carried; they were emaciated and cyanotic, the area of cardiac dulness was increased, and souffles were present; yet these same children would return and be shown to the next class of students in the following year, walking up-stairs without dyspnoea, looking well nourished, of good color, with much less enlargement of the area of cardiac dulness, and with the cardiac souffle scarcely perceptible, showing that the cardiac compensation was complete.

As an illustration of this class of cases you will perhaps remember the little girl (Case 495) who was brought to the clinic by her mother to be shown as a child who was then well, but whose chances of living had seemed at one time very slight.

When first seen she was about five years old. She had never had any of the acute diseases, such as scarlet fever, diphtheria, pertussis, articular rheumatism, or in fact any disturbance except slight pains in her limbs. For the previous six months she had lost in appetite and weight, got out of breath very easily, suffered from palpitation, and in the beginning of her sickness was confined to her bed for a week or ten days with a high fever and pain referred to her left side. On examination she was found to be somewhat cyanotic. The area of visible cardiac pulsation was much increased. The apex of the heart was in the sixth interspace, 3 cm. ( $1\frac{1}{8}$  inches) to the left of the mammary line. The area of absolute cardiac dulness extended to the right parasternal line, from the third to the fifth cartilage, and 1 cm. ( $\frac{3}{8}$  inch) to the left of the mammary line on a level with the left nipple; the vertical area of dulness to the left of the sternum extended from the second to the sixth interspace. There was a loud mitral systolic murmur. The lungs were normal.

The chief points of treatment in this case were the careful administration of nourishing food and the enforcement of rest. She was always carried up and down stairs for almost a year. She grew worse for a time; she became irritable, and for some time when the cyanosis and orthopnoea were most marked she had a cough, and once or twice hæmoptysis. By the following winter, however, the general symptoms were much improved, and in another year the dyspnoea, cyanosis, palpitation, and pain had passed away. The apex of the heart was found to be in the fifth interspace in the mammary line, and the area of dulness very little greater than normal.

Cardiac symptoms dependent on organic lesions may arise, and yet no physical signs of such lesions be detected during life.



**Mechanical.**—The mechanical conditions in cardiac disease play a very interesting and important part in many diseases in infancy and early childhood, and by their results often interfere seriously with the general physical condition and normal development of the child. These abnormal conditions may result in hypertrophy or dilatation from over-exertion; they may occur at puberty; they may arise from direct mechanical interference with the heart's action, as from adhesions or from undue pressure on the cardiac cavities, as in pulmonary disease, pertussis, renal disease, and narrowing of the aorta. In all these diseases there is a greater liability that acute dilatation may take place in early life than that it may occur at a later period. You should therefore always remember to examine the heart carefully during the course of all these diseases. The processes which suddenly cause great increase of the blood-pressure in the lungs may lead to acute dilatation of the right ventricle, while where there is a diffuse renal disease, as in scarlet fever, acute dilatation of the left ventricle may take place, and be followed by hypertrophy, as I have explained to you in my lecture on scarlet fever (page 569, Case 245). In all these diseases this acute dilatation may take place rapidly and disappear almost as rapidly, a phenomenon which is somewhat characteristic of cardiac disease in early life.

I have already referred to the great changes which take place in the heart, and to its rapid growth, at the time of puberty. At this period the general growth of the child is apt to be very rapid, and symptoms of cardiac weakness commonly occur, especially in girls. These symptoms are debility, lack of energy, palpitation, and dyspnoea on exertion. There may also be signs of slight cardiac dilatation, and murmurs, probably hæmic in their nature. This period, therefore, is one in which cardiac disease from any cause, such as rheumatism, is of more serious import than at a later period, when the heart is not taxed by too rapid growth.

These cases should be treated by mild physical exercise, care being taken that the children do not over-exert themselves. Complete rest for two or three hours every day should be enforced. Under this treatment, combined with nutritious food and possibly a tonic of iron or nux vomica, the signs of cardiac disturbance usually soon disappear.

I must again remind you of the importance of mechanical interference with the action of the heart arising from adhesions. Adhesions of the pericardium or in its neighborhood are so latent in infancy in their symptoms that they are often overlooked until the mechanism of the heart has become so seriously interfered with as to present the symptoms of disease of the heart itself, such as dilatation or hypertrophy.

**Inflammatory.**—The chief inflammatory lesions of the heart are *endocarditis* and *myocarditis*.

**Endocarditis.**—The most common cardiac disease which occurs in children is endocarditis. Endocarditis may be acute or chronic, primary or secondary.

ETIOLOGY.—The elaborate investigations of J. H. Wright, W. R. Stokes, and others have shown that acute endocarditis is of bacterial origin. Weichselbaum has contributed more to our knowledge of this disease than any other investigator. He has proved that there is no essential difference between the various forms of endocarditis, either histologically or pathologically, and that no one species of bacteria is exclusively concerned in the production of the disease. Sometimes the streptococcus pyogenes is found, sometimes the staphylococcus pyogenes aureus, and sometimes the diplococcus pneumoniae. We therefore no longer need make a distinction between simple endocarditis and ulcerative or verrucose endocarditis. There is merely a difference in the degree of the malignant nature of the especial organism which has produced the disease, or in the vulnerability to infection of the individual.

PATHOLOGY.—While the same lesions of endocarditis may be found in children as in adults, yet in infancy, although marked acute cardiac symptoms and murmurs frequently arise, the autopsy almost invariably fails to show any endocardial lesions or growths. In two thousand autopsies at the New York Foundling Asylum, Dr. Northrup and Dr. O'Dwyer never found an acute inflammatory lesion except in one case, which showed the lesions of acute malignant endocarditis. Where the lesions of endocarditis are found in children, the connective tissue and the basement substance are, according to Delafield and Prudden, principally concerned in the inflammatory process. The endocardium which forms the valves is that which is most frequently inflamed, but other portions of it are by no means exempt. In some cases there is swelling of the valves, which are thickened, their surfaces remaining smooth, the basement substance is swollen, and there is a moderate production of new connective-tissue cells. In other cases the growth of connective-tissue cells is very much more marked, the basement substance is broken up, and little cellular fungus-masses, called vegetations, project from the free surface of the endocardium. In still other cases the cellular growth in some places forms vegetations, and in others degenerates, and thus portions of the valves are destroyed. This is *simple acute ulcerative endocarditis*.

In some cases the children recover, and the valves seem to return to their normal condition, while in others the valves are left permanently damaged.

*Chronic endocarditis* may succeed an acute endocarditis, or the inflammation may be chronic from the onset. It affects most frequently the aortic and mitral valves and the endocardium of the left auricle and left ventricle, similar changes in the right side of the heart being much less frequent. In these cases the endocardium may be thickened and tense, and its surfaces smooth or covered with small, hard vegetations or ridges, or there may be a growth of connective-tissue cells in the endocardium, with a splitting up of the basement substance.

While endocarditis may be primary, simply arising from the infection



of some organism, it is commonly secondary. It arises most frequently in connection with rheumatism and chorea, also in the course of the acute exanthemata, especially scarlet fever, and in diphtheria. Acute endocarditis may also be secondary to old cardiac malformations or lesions (*endocarditis recurrens*).

In connection with endocarditis *myocarditis* may be present. In this event there is an inflammatory change in the walls of the heart, involving primarily the interstitial tissue and blood-vessels, the muscular fibres being secondarily affected by atrophic and degenerative changes.

SYMPTOMS.—The symptoms of *endocarditis* are often obscure, and in infants and young children, in the beginning, are apt to be latent. When the disease arises in connection with some other disease, such as rheumatism, the symptoms are especially likely to be masked by those of the disease which it complicates. In some cases the endocarditis develops insidiously without any additional symptoms, and its presence is not recognized until a careful examination of the heart detects a murmur; in others pronounced and even violent cardiac symptoms are present from the beginning. If the muscular tissue is involved as well as the endocardium, the general cardiac symptoms of dyspnoea, cyanosis, and palpitation are still more marked.

The symptoms of *myocarditis*, however, are so closely associated with those of an accompanying endocarditis or pericarditis that clinically, as a rule, they cannot be separated from them.

Where endocarditis does not arise as a complication of some other disease, the symptoms at the onset, when prominent, are usually a rise of temperature, a quickened and sometimes weak and irregular pulse, dyspnoea, palpitation, and more or less præcordial distress. All these symptoms vary according to the extent of the lesions. Later they depend upon whether or not compensation has been established. In connection with these early symptoms, cardiac dilatation and cyanosis are very apt to occur. When the disease has advanced far enough to cripple the heart and to interfere with compensation, the physical signs of enlargement appear, such as increase in the area of cardiac dulness and the presence of murmurs corresponding to the orifices affected. The symptoms differ somewhat according as the inflammatory condition has begun in the valves or in the cardiac walls. (Steffen.) In the former case the signs of dilatation accompany those of valvular weakness, while in the latter the symptoms of dilatation come first, and are followed by the mechanical results of valvular insufficiency.

In a first attack of acute endocarditis such serious symptoms connected with great lack of compensation as are met with where the attack supervenes on a previous cardiac lesion are not likely to arise. In some cases, however, where the individual power of cardiac resistance is slight, these advanced symptoms appear. Under these circumstances the child emaciates rapidly, becomes very weak and anæmic, and the cyanosis and dyspnoea,

the latter of which may amount to orthopnoea, increase. There is apt to be cough from an accompanying bronchial irritation, produced most frequently where there is obstruction at the mitral orifice, and, following a general venous stasis, enlargement of the liver, hæmoptysis, and œdema of the face, legs, and arms appear. Children show such a wonderful recuperative power that even in these advanced cases under proper treatment the serious symptoms may gradually pass away, and often such complete cardiac compensation takes place that they are left with no symptoms of cardiac disease except a murmur.

In endocarditis relapses are common and there is a great tendency to recurrence. Embolism may take place, and sometimes the first symptom of cardiac disease which has been noticed is a hemiplegia following a lesion of the mitral valve. Anæmia is a very common symptom, especially where endocarditis accompanies rheumatism. Congestion of the lungs, with resulting hæmoptysis, may arise where there is insufficiency of the mitral valve. Although when the valves are affected murmurs are usually present, yet sometimes where there are lesions of the valves murmurs cannot be detected. In endocarditis murmurs are most frequently heard in the region of the mitral valve, and insufficiency of the mitral valve is the most common of the inflammatory cardiac lesions in childhood.

DIAGNOSIS.—The diagnosis of endocarditis depends upon the physical signs. These signs are an increase in the area of cardiac dulness and a change in the cardiac sounds. The change in the area of cardiac dulness must be differentiated from that which occurs in a pericardial effusion, of which I shall speak later (page 1056).

The change in the cardiac sounds may be produced by changes in the blood or by organic lesions of the valves. The differential diagnosis between these two conditions is the same as in adults, and therefore I shall not dwell upon it. In insufficiency of the mitral valve the murmur is systolic, and is transmitted to the axilla and the back. In some cases the murmur of mitral insufficiency is closely simulated by a valvular sound produced in the course of pericarditis. Stenosis of the mitral valve is much less common than insufficiency. It is represented by a presystolic murmur heard in a limited area in the region of the heart's apex, and is sometimes accompanied by a reduplication of the cardiac sounds at the apex and by a thrill. Pain is said to be more common in connection with this lesion than with other cardiac defects. Lesions of the *aortic valve* are almost invariably associated with rheumatism. Stenosis of the aortic orifice is very apt to be associated with a mitral lesion. There is nothing especially characteristic in childhood of these lesions of the aortic valves, and the same may be said of lesions of the tricuspid valves.

Where endocarditis has become chronic and compensation has only partially taken place, the children are atrophic, anæmic, and have a tendency to imperfect circulation and to bronchitis. In some cases the fingers become club-shaped.



PROGNOSIS.—The prognosis of acquired endocarditis in early life is comparatively favorable. I have already referred to the great recuperative powers of the child, and in many cases, especially where it is the first attack, such complete compensation takes place that the child practically recovers. If it is the walls of the heart that are affected, the heart may regain its normal size and position. If the valves alone, or the valves and the walls, are affected, reaction can still take place. Death may, however, occur at the height of the attack, or the child may die later from exhaustion and sometimes suddenly from heart-failure.

TREATMENT.—The treatment of acute endocarditis during the early days of the attack is essentially rest in bed, and is otherwise symptomatic. From the very beginning, however, we must bear in mind that our treatment should be directed to establishing compensation. We should also remember that the younger the child the more likely it is that we shall have to contend with a resulting atrophic condition and anæmia. The child should be encouraged to sleep, in order that the circulation may be kept as quiet as possible and thus relieve the work of the disabled heart. The heart-beats of a young child during sleep are often reduced twenty in a minute, and thus sleep affords the best opportunity for compensation. The treatment which I have found most efficient in all forms of cardiac disease is absolute rest in bed for days or even weeks until compensation has become complete. Later the general health of the child should be carefully attended to by means of good food, pure air, and exercise of a mild type, never excessive. The surface circulation should be promoted by baths and gentle massage. Digitalis and iron are of great value, the former in aiding the establishment of compensation, the latter in combating the anæmia. If at any time during the course of the disease the attacks of dyspnoea are excessive, nitroglycerin can be given in doses proportionate to the age of the child; 0.0003 gramme ( $\frac{1}{2000}$  grain) can be given to a child three or four years old.

Although the more advanced pathological condition usually spoken of as “ulcerative endocarditis” rarely occurs in young children, yet it is at times met with. Its symptoms are obscure, and the diagnosis is rarely made during life.

I have here the organs of a child who has lately died in my wards with this disease.

This boy (Case 496), four years old, was attacked one month ago with fever, thirst, and pain in his knees. Later his feet became painful and swollen, and other joints were successively involved. He complained of pain in the back of his neck and along his spine. One week before entering the hospital he began to have moderate but incessant choreic movements, and he showed much incoördination of mastication and articulation.

A physical examination showed the lungs to be normal, the area of cardiac dulness somewhat increased to the left of the mammary line, and a murmur at the apex, with the first sound transmitted to the axilla and the back. On the following day a pericardial friction-sound was heard just above the left nipple, accompanied by præcordial pain. Two weeks later the choreic symptoms disappeared, and the temperature became normal. The

area of cardiac dulness did not extend under the sternum, but was found to correspond to the impulse of the heart, which was 1.4 cm. ( $\frac{1}{2}$  inch) outside of the left mammary line. During the last week of its life the child became very weak, had marked dyspnoea, and showed signs of effusion in the right pleural cavity, but presented no other symptoms. It died yesterday.

The post-mortem examination was made by Dr. Councilman.

Both pleural cavities contained a considerable accumulation of blood-stained fluid. The anterior mediastinum was deeply injected and reddened, and the mediastinal lymph-glands beneath the sternum were enlarged. The apex of the pericardium was tightly adherent to the left pleura, and about this point the tissues were thickened, deeply injected, and oedematous. The right lung was adherent to the pleura by comparatively fresh adhesions. Here and there over the pleural surface of the lung is a slight fibrinous exudation. The lymphatics over the surface of the pleura are greatly dilated. The upper lobe of the right lung is congested. Small nodular masses can be found beneath the pleura, and on section, as you see, there is a distinct lobular consolidation throughout the upper lobe of the right lung. The solid portion of the lung is of a dark-red color and comparatively smooth on section. Muco-purulent matter can be squeezed from the larger bronchi. The small consolidated areas are more or less separated from one another, and between them are cavities in the interlobular septa. The appearance of the lung is somewhat similar to that presented in bovine pleuro-pneumonia. The bronchial glands are enlarged and reddened. The left lung was not so adherent as the right. All over the posterior portion of the pleura there was a slight fibrinous exudation. This lung has been somewhat compressed by the accumulation of fluid in the pleural cavity, but otherwise shows about the same condition as the right lung, the consolidation being in the posterior portions principally. The pleural cavity, as you see, is obliterated by the adhesions. The parietal pericardium is greatly thickened, and in and between the connective-tissue adhesions there is a thick fibrinous exudation. The heart is somewhat enlarged. At the apex of the left ventricle, at a point corresponding to the adhesions of the pericardium, the myocardium feels soft and is somewhat whiter than the remainder of the tissue. The interior of the right heart contains tolerably firm, fresh clots. The myocardium of the right side of the heart is pale and soft. Along the free border of the right auriculo-ventricular valve there are a few fresh vegetations. The left side of the heart is dilated. The edge of the mitral valve is thickened and eroded. There appears to be a slight loss of substance in the thickened portion of the valve, and the edges are irregular and eroded. The muscular substance of the heart is generally pale. Beneath the endocardium there are small, whitish points. Similar points are seen on the papillary muscles and on the inner side of the auricle. The aortic valves are intact, except for a few fibrinous deposits just at the edges of contact. The coronary arteries are normal.

The spleen is enlarged and comparatively soft. The mesenteric lymph-glands are enlarged and also slightly soft. The liver is large, dark red in color, and the lobules are prominent.

The left jugular vein is filled by a firm, adherent thrombus, which extends downward into the subclavian vein, the innominate, and the superior vena cava, and completely obliterates those veins.

A microscopic examination of the lungs shows a distinct lobular pneumonia. The alveoli contain very little fibrin, but are filled with large, pale cells. Among these are a few leucocytes, but usually the leucocytes are conspicuously absent. The consolidation is quite general, comparatively few of the alveoli in the most affected portions being free. At numerous places in the lung there are wide passages, apparently lymphatics, filled with fibrin and large, pale cells similar to those in the alveoli. The bronchi are in most cases free. The lung consolidation does not appear to take its point of departure from the bronchi.

Typical masses of streptococci are found in the alveolar contents and in their walls. The lymphatics of the pleura are enlarged, and correspond to the large passages just described in the lung. Sections of the bronchial and cervical lymph-glands show acute swelling of the glands, with micrococci here and there in the sinuses.

A microscopic examination of the heart showed the vegetations on the mitral valve to



be distinctly verrucose. Here and there on the ends of these vegetations were small masses of fibrin. Only in places was there a direct infiltration with leucocytes. Streptococci were found on the edges of the vegetations, chiefly in the fibrin. In but one place were they found within the tissue. Sections of the myocardium embracing the pericardium showed a fibrino-purulent exudation on the pericardial surface. Numbers of streptococci were found in the fibrinous exudation and in a few places on the edge of the cardiac muscle. Sections from the left ventricle showed a marked adhesion with the pleura and an acute inflammation with a few streptococci in the tissue. The thrombi in the large cavity showed no evidence of organization, and no streptococci were found in them, but there were numbers of them in the perivascular tissue, which showed slight purulent infiltration.

Sections of the liver, kidney, and spleen showed no pathological condition save a slight, cloudy swelling, and no organisms were found in these tissues.

Cultures made at the autopsy gave a pure culture of streptococci from the lungs, from the pericardium, and from the bronchial lymph-glands. In the spleen only a few colonies were found. The other organs were sterile.

#### CASE 497.



Acute endocarditis. Mitral insufficiency. Male, 8½ years old.

As Dr. Councilman says, the most interesting part of the autopsy is the manner of infection. He thinks that the heart must have become infected before the lungs, so that apparently this is a case of primary endocarditis of the malignant form. It seems very probable to Dr. Councilman that the path of the infection was from the heart to the pericardium, thence to the mediastinum, producing the thrombus of the veins, and probably thence to the lung, possibly by means of the thrombosed veins. The thrombi seem to have been due to an inflammation of the wall of the vein, produced by the streptococci in the perivascular tissue. From this point they could have got into the veins, the infection being carried thence into the lungs. The pneumonia in the lungs is entirely different from the ordinary broncho pneumonia of infants, which is due to aspiration, and in which the chief seat of the disease is in the bronchi and the surrounding lung-tissue. In this case, however, the bronchi are less involved than other portions of the lung.

I have here in the wards a number of cases which illustrate the various types of cardiac disease.

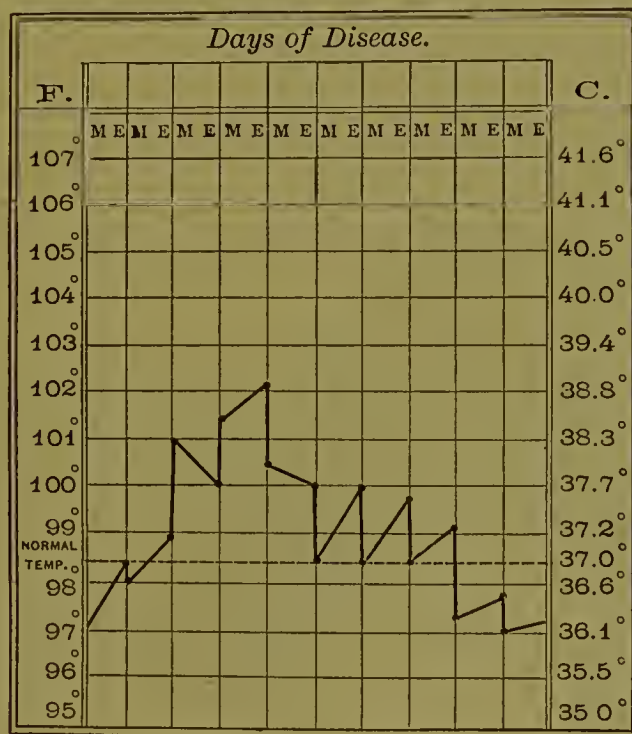
This boy (Case 497, page 1036), eight and a half years old, was well until nine weeks ago. He had never had any diseases, except measles and variella when he was four years old. Nine weeks ago he was attacked with chorea, which lasted for about seven weeks and was succeeded by symptoms of dyspnœa on exertion, loss of appetite, and slight cough. There has been no history of rheumatism in the case. You see that he is cyanotic, but otherwise looks comparatively well.

There is no œdema, and an examination of the lungs detects nothing abnormal. The impulse of the heart is in the left mammary line in the fifth interspace. The cardiac area of absolute dulness is as I have indicated with this black curved line. I have also marked the lower border of the ribs with a plain black line, and have shown the slightly enlarged liver and spleen with a broken line. The dulness does not extend beyond the middle of the sternum, but is increased in the vertical line as high as the second interspace and extends slightly beyond the left mammary line. There is a marked systolic murmur, heard most loudly at the apex, and transmitted to the axilla and the back, also to the base of the heart.

This appears to be a case of acute endocarditis arising during an attack of chorea. The prognosis is good, as the child is already improving.

(Subsequent history.) The child was treated simply by rest in bed, and a month later his general symptoms improved, the areas of splenic, hepatic, and cardiac dulness were much decreased, and the cardiac murmur was not so distinct. Two weeks later compensation was apparently established, he had gained in weight, his color became better, and he left the hospital in good condition.

CHART 45. (CASE 498.)



Acute endocarditis. Female, 9 years old.

I have here a girl (Case 498, I., page 1038), nine years old, who, although she has always been a delicate child, never had any especial disease until two weeks ago, when she was attacked with fever, palpitation, cough, and a rapid, irregular pulse. On entering the hospital she was cyanotic, the face and extremities were cold, and there was considerable prominence over the cardiac region. The resonance of the lungs was normal, but there were a few moist râles at both bases. The impulse of the heart was in the fifth left interspace, 1.4 cm. ( $\frac{1}{2}$  inch) outside of the mammary line, and there was a marked thrill with a systolic murmur transmitted to the axilla and heard distinctly in the back. The liver was slightly enlarged. Here is the temperature chart (Chart 45), showing the temperature during the acute inflammatory stage of the endocarditis.



The impulse of the heart is scarcely perceptible. The area of cardiac dulness extends, as you see, to the right edge of the sternum, and slightly beyond the right parasternal line beneath the third intercostal space.

## CASE 498.

## I.



Acute endocarditis. Mitral insufficiency. Lack of compensation. Orthopnea. Female, 9 years old.

The case illustrates an attack of acute apparently primary endocarditis. The acute inflammatory stage has been passed; dilatation has taken place, and there is at present marked failure of compensation. This is shown by the feeble impulse of the heart, the weak and fluttering pulse, the cold and blue extremities, the orthopnea, and the tendency to edema of the face, legs, and feet. You see the position which the child assumes on her right side, how she supports herself with her arms, and her anxious expression as she endeavors to keep herself in a position in which she can breathe easily.

## CASE 498.

## II.



Acute endocarditis. Dilated heart. Orthopnea. Position assumed when sleeping. Female, 9 years old.

A case of this kind needs to be very closely watched, as the child is liable to die suddenly. A few days ago, in this next bed, there was a boy (Case 499), four and a half years old, who was suffering from an attack of acute endocarditis: he had the same symp-

toms of dilated heart with a lack of compensation as you see in this little girl. While he was sleeping in the same position which she has assumed since I have been describing her case to you (Case 498, II., page 1038), he died suddenly. He had been subject to sudden violent attacks of dyspnœa, and once or twice while in the hospital had an attack of angina pectoris.

This next child (Case 500), a girl, thirteen years old, has a history of pertussis some years ago, but has not had any other disease except an attack of rheumatism two years ago. Since then she has occasionally had attacks of dyspnœa when at play and when going upstairs. She has also at times had œdema of the feet. One week ago she complained of pain in the cardiac region, so severe as to interfere with her sleep. On entering the hospital she had a temperature of  $38.5^{\circ}$  C. ( $101.2^{\circ}$  F.), a pulse of 104, and respirations 65. An examination showed nothing abnormal except in the cardiac region.

CASE 500.



Chronic endocarditis following rheumatism. Mitral stenosis and insufficiency. Anæmia. Female, 13 years old.

The impulse of the heart is in the fifth left interspace in the mammary line. The area of absolute cardiac dulness is enlarged, and I have represented it by a black curved line. You see that it extends beneath the sternum, and at the junction of the upper border of the fourth rib extends a short distance to the right of the sternum. The upper boundary, as I have said, is the upper border of the third rib, and is about 5.3 cm. (2 inches) outside of the mammary line. There is a presystolic murmur at the apex, which is confined to a limited area. There is also a systolic murmur at the apex transmitted to the axilla and the back; the pulmonic second sound is accentuated.

(Subsequent history.) Four weeks later, after being treated by complete rest in bed, the cardiac symptoms almost entirely disappeared, the area of cardiac dulness was much diminished, and the murmurs were less distinct. Two weeks later the child left the hospital, much improved in her general health, but in a very anæmic condition.



This next boy (Case 501), nine years old, had an attack of rheumatism when he was six years old.

## CASE 501.



Chronic endocarditis following rheumatism. Mitral insufficiency. Pericarditis sicca. Dilated heart. Pneumonia. Pleurisy. Male, 9 years old.

Four weeks before entering the hospital he began to have swelling of the feet, and four days before entrance swelling and pain in the cardiac region and much dyspnoea and general discomfort. His respirations and pulse were much quickened, and his temperature was raised. On entering the hospital, a physical examination showed that he had pneumonia of the left lung. A pericardial friction-sound was also heard in the second left interspace. The cardiac area of absolute dulness extended as far as the right parasternal line, as high as the third rib, and 5.3 cm. (2 inches) beyond the left mammary line. There was a soft systolic murmur at the apex. The pneumonia involved the whole of the left lung, and was complicated by a moderate pleuritic effusion. Resolution took place, however, and the fluid was absorbed. The cardiac symptoms improved as soon as the pneumonia and pleurisy disappeared, but the physical signs of the dilated heart have not yet changed. For some weeks I shall enforce absolute rest in bed, as this morning he was suddenly attacked with extreme dyspnoea, cyanosis, and collapse, which followed his getting out of bed and dressing himself contrary to my directions.

I have indicated the area of absolute cardiac dulness in black, which shows an enlarged heart, as you will understand better when I describe the dulness produced by a pericardial effusion. There is no visible impulse of the heart, and the beat can scarcely be found on palpation. The increased area of dulness is therefore practically caused by dilatation rather than by hypertrophy, and this supposition is substantiated by the symptoms of lack of compensation which he has shown.

This boy (Case 502, page 1041), eleven years old, had measles when he was an infant, diphtheria when he was three years old, and pertussis when he was four years old. He had always been well until one and a half years ago, when, after indefinite pains in his joints, accompanied by no swelling and not sufficiently severe to confine him to bed, he began to have dyspnoea on exertion, and cardiac pain. He is somewhat cyanotic, and has lately lost a great deal in weight. There is no oedema, and nothing else abnormal is detected except in the examination of the heart, which shows the area of absolute dulness to be somewhat increased. A loud presystolic murmur is heard at the apex, limited in its extent and accom-

panied by a thrill. He has also had a cough. He seems to represent a case of stenosis of the mitral valve. There is, as you see, decided enlargement in the cardiac region to the left of the sternum.

(Subsequent history.) After remaining in the hospital for two months and being treated by rest, compensation was established, and he left the hospital in good condition.

CASE 502.



Chronic endocarditis. Mitral stenosis. Bulging of left side of sternum. Male, 11 years old.

Since then he has returned from time to time with a renewal of the symptoms of cyanosis, dyspnoea, and lack of compensation.

This next boy (Case 503, I., page 1042), ten years old, is interesting as illustrating certain characteristics of cardiac disease in early life.

Two years ago he entered the hospital with marked œdema of the face, body, and limbs, ascites, a slight amount of fluid in both pleural cavities, and œdema of the lungs. There was no definite history of rheumatism nor any other cause for the cardiac disease which was causing these symptoms, and which had apparently developed insidiously, though if he had been under closer observation a definite period of onset would probably have been discovered. The impulse of the heart was found to be 1.4 cm. ( $\frac{1}{2}$  inch) outside of the mammary line in the fifth left interspace. The area of cardiac dulness was somewhat increased. There was a loud systolic murmur at the cardiac apex transmitted to the axilla. The second pulmonie sound was much accentuated. Here is a picture (Case 503, II., facing page 1042) taken at that time, and showing the marked œdema of the legs and the much distended abdomen. He was treated by complete rest in bed for five weeks, and in the beginning digitalis was administered until the urine, which was lessened in quantity, had increased and the œdema of the lungs had disappeared. On entering the hospital the ascites was removed by paracentesis abdominis. Under this treatment the child rapidly improved, the general œdema disappeared, the liver returned to its normal size, the area of cardiac dulness was markedly decreased, the cardiac murmur became less marked, and six weeks from the time when he entered the hospital complete compensation was established and he left the hospital seemingly perfectly well. This picture (Case 503, III., facing page 1042),



was taken just before he left the hospital, and, as you see, is in marked contrast to the picture taken on his entrance.

## CASE 503.

I.



Chronic recurrent endocarditis. Mitral insufficiency. Disturbance of compensation. Dilated heart. Enlarged liver. Œdema of lungs. Ascites. Male, 10 years old.

Since leaving the hospital the boy is reported to have been very well, except that he could not play or work hard. Two weeks ago he was attacked with fever, præcordial distress, and cardiac pain; later he began to have œdema of the feet and dyspnœa. Since then he has been growing progressively worse, and his case illustrates a fresh attack of endocarditis supervening on an old chronic endocarditis (endocarditis recurrens) and resulting in a disturbance of the previous compensation. You see that he has orthopnœa to such an extent that he is unable to lie down in bed, and that he has to be continually watched by a nurse, as he frequently has attacks of excessive paroxysmal dyspnœa which are liable to prove fatal. There are cyanosis of the lips and hands and marked general œdema. The skin of the nose and extremities is cold. The impulse of the heart is felt in the sixth left interspace 2.8 em. (1 inch) beyond the mammary line. The area of cardiac dulness extends beneath the sternum, and at the third intercostal space extends 1.4 em. ( $\frac{1}{2}$  inch) to the right of the sternum, thence upward in a curved line across the upper part of the sternum to the second rib, and then, keeping outside of the mammary line, descends and joins the point of cardiac impulse. There is a loud systolic murmur, heard most distinctly at the apex, but transmitted over the whole cardiac area and through the axilla to the back. The second pulmonic sound is accentuated. The aortic sounds are weak. There are numerous moist râles heard in all parts of the lungs. The percussion of the lungs is resonant everywhere except in the lower parts, where there seems to be a slight amount of fluid in both

CASE 503.

II.



Chronic endocarditis. Mitral insufficiency. General œdema and ascites. (Before treatment.)

CASE 503.

III.



Chronic endocarditis. Complete compensation. (Six weeks after treatment.) Male, 10 years old.





pleural cavities. The liver is enlarged so that it extends 7.8 cm. (3 inches) below the margin of the ribs. Ascites is present, the fluid rising to about the line of the umbilicus. The spleen is normal in size. The child is passing only a small amount of urine, which contains a trace of albumin. I have marked the cardiac and hepatic areas of dulness and the upper border of the ascites by black lines, the margin of the ribs by broken lines, the point of cardiac impulse by a black ring, and the œdematous râles in the chest by smaller black rings. The prognosis in this case, although from the child's present condition very serious, as he is liable to die suddenly at any time if extra blood-pressure should be brought to bear upon the dilated and crippled heart, is not entirely unfavorable, as he has previously shown such great powers of compensation and recuperation. As there is no great distention of the abdomen, I shall not at present remove the ascites by paracentesis, but shall have the child carefully watched, and, if the ascites increases, shall have it removed at once. He is taking infusion of digitalis, 3.75 c.c. (1 drachm), every three hours, and diuretin, 0.36 gramme (6 grains), once in six hours as a diuretic. His diet is milk.

(Subsequent history.) Within forty-eight hours rapid relief was obtained from the urgent symptoms, and at the end of three weeks the œdema of the lungs, the general œdema, and the ascites had disappeared entirely. The urine became normal in quantity and free from albumin. One week later he was well enough to be out of bed for an hour each day, and at that time this picture was taken (Case 503, IV.), which shows the heart and liver to be still enlarged.

## CASE 503.

IV.



Chronic endocarditis. Mitral insufficiency. Returning compensation. Enlarged liver. Enlarged heart.

V.



Chronic endocarditis. Mitral insufficiency. Broken line indicates enlarged heart. Black line indicates area of cardiac dulness with returned and complete compensation.

Some weeks later the liver regained its normal size, and still later the cardiac area of dulness was found to be much reduced and in the vertical line almost normal. This picture (Case 503, V.) shows the enlarged heart, which remained longer than the enlarged liver,



and is represented by a broken line; the area of dulness of the heart as it appeared when he left the hospital is shown by a black curved line.

This next case, a girl (Case 504), nine years old, is instructive as showing the difference between the cardiac area of dulness produced by an enlarged heart and that produced by a distended pericardium. She had pertussis when she was three years old, and measles when she was seven years old. This was followed by an attack of rheumatic fever, which lasted six weeks. So far as I can ascertain, she had no cardiac disturbance at that time, and recovered completely from the attack of rheumatism. Two months later she had another attack of rheumatism, which was accompanied by pain in the cardiac region. She then apparently recovered, but one year later had a recurrence of the cardiac disturbance, which was, however, of short duration. From that time she remained well until four months ago, when she had a severe attack of bronchitis, and since then she has been failing in strength and has suffered from dyspnoea. Three weeks ago she began to have œdema of the feet and of the abdomen, and this has been progressively increasing. She is very anæmic, and, as you see (Case 504, I., facing page 1044), the œdema of the face and legs is marked. The distention of the abdomen is found to be produced by ascites. An examination of the heart shows the apex-beat to be in the sixth interspace, 4 cm. ( $1\frac{1}{2}$  inches) beyond the mammary line. The area of absolute dulness extends from the third left costal cartilage downward across the sternum to 2.8 cm. (1 inch) beyond the right parasternal line in the fifth interspace. It also extends to the left and downward outside the mammary line until it joins the impulse of the heart in the sixth interspace. This area of dulness is not that which we meet with in a dilated heart alone, as I shall presently explain to you. On the contrary, it suggests that there is fluid in the pericardium.

In connection with the general œdema and absence of symptoms of pericarditis there is probably present the condition called hydropericardium. The liver is also enlarged. The pulse is regular, 140. There is a systolic murmur at the apex of the heart.

She is being treated by absolute rest in bed, a milk diet, infusion of digitalis, and diuretin.

(Subsequent history.) After she had been in the hospital for forty-eight hours the hydropericardium disappeared, the skin became less tense, the urine increased in amount,

#### CASE 505.



Chronic endocarditis. Greatly dilated heart. General œdema. Extreme distention of abdomen with ascites. Female, 11 years old.

and there was rapid improvement in all the general symptoms. Two weeks later the œdema and ascites disappeared entirely, as is shown in a picture (Case 504, II., facing page 1044) taken at that time. The enlarged heart at that time is indicated by a broken line, while

CASE 504.

I.



Chronic endocarditis. Mitral insufficiency. Hydropericardium. General cedema and ascites. The line of ascites and of the cardiac area of dulness marked in black. (Before treatment.)

CASE 504.

II.



Chronic endocarditis. Enlarged heart. Emaciation. (Two weeks after treatment.) Female, 9 years old.





the area of dulness, which was found some weeks later when compensation was established, is represented by a black line. The child was left in a very anæmic and emaciated condition, but the liver resumed its normal size, the area of cardiac dulness gradually became smaller, compensation was finally established, and she left the hospital in good condition.

Here is a girl (Case 505, page 1044), eleven years old, who has just been admitted to the hospital.

She shows, as you see, extreme dyspnœa, orthopnœa, cyanosis, marked general œdema, and great distention of the abdomen produced by ascites. Percussion of the chest shows extreme dilatation of the heart over an area which includes the entire sternum from the second interspace and extends 7.8 cm. (3 inches) to the left of the mammary line, the impulse of the heart being in the seventh interspace. There is also extensive œdema of the lungs. The pulse is weak and feeble. It is very evident that there is an entire lack of compensation in this case, and that, unless the heart is quickly relieved, cardiac failure will take place and the child will die. I have therefore told the mother that paracentesis abdominis must be performed at once.

(Subsequent history.) The mother refused to have paracentesis performed, and took the child home: it died suddenly on the following day from heart-failure.

I have already referred to the deformities which may arise in the chest from the pressure of an enlarged heart during a period when the thoracic walls are still pliant and undeveloped.

Here is a little girl (Case 506) who six years ago had an attack of rheumatism followed by endocarditis, and, although compensation has taken place and she is fairly well and strong, you see the displacement of the sternum and of the costal ends of the left ribs which has resulted from the cardiac enlargement.

CASE 506.



Displaced sternum and costal cartilages from enlarged heart. Female, 10 years old.

CASE 507.



Malformation of left side of thorax from cardiac disease.

Here is another case (Case 507) of cardiac disease, in which the endocarditis with its resulting cardiac dilatation and hypertrophy occurred at a still earlier period of life, and, as you see, there is great deformity of the left side of the thorax produced by the intrathoracic pressure.



## LECTURE LII.

## DISEASES OF THE PERICARDIUM.

THE anatomy of the infant's pericardium, so far as I have been able to determine by the dissection of sixteen infants of various ages, appears to approximate so closely that of the adult that there is nothing distinctive to note concerning it. The amount of fluid which normally occurs in an infant's pericardium, although of variable quantity, is probably under 5 c.c.

The chief diseases which affect the pericardium are hydropericardium, hæmopericardium, pneumopericardium, and pericarditis. The first three are very rare in early life, and therefore need be merely referred to. Absence of the pericardium may occur, and may be complete or partial.

**PERICARDITIS.**—The most common disease of the pericardium is pericarditis. It can occur at all ages, but the earlier the age the less often is it met with. It has been found in the foetus and in the new-born, and well-marked adhesions of the pericardial surfaces have been observed in an infant which died thirty-six hours after birth.

**ETIOLOGY.**—There are a number of organisms which seemingly give rise to pericarditis. The most common of these is the micrococcus lanceolatus. In the new-born pericarditis may be the result of a septic condition following infection of the cord. At times it follows periostitis and otitis in young children, here also probably being associated with septic infection. Traumatism may also be a cause of pericarditis. Rheumatism, especially after the third or fourth year of life, gives rise to as much periendocardial disease as at a later period. The inflammatory lesions may arise before the rheumatism has appeared elsewhere, and the intensity of the arthritic pain and the number of joints affected do not correspond to, or rather do not influence, the frequency of the pericardial complication. Inflammation of the pericardium is also frequently associated with pneumonia. It may be secondary to any of the eruptive fevers, but occurs most frequently as a complication of scarlet fever. When it occurs in this latter disease it appears usually in the second or third week of the attack. The pericardium also shows an especial tendency to invasion by the bacillus tuberculosis following tuberculosis of the pleura.

**PATHOLOGY.**—Pericarditis may be circumscribed or diffuse, and there appears to be no essential difference between the pathological conditions affecting the pericardium in early life and those which occur later. The pericarditis sicca of the adult is comparatively unusual in the child, in whom, as a rule, an effusion of greater or less extent almost always takes place. The effusion may be sero-fibrinous, hemorrhagic, or purulent. Not only is the tendency to effusion in the child greater than in the adult, but its forma-

tion is characterized by greater rapidity and it is more likely to be purulent than in the adult. A pericardial effusion tinged with blood is not uncommon in early life, and is not necessarily so significant of tuberculosis as is a pronounced hemorrhagic effusion. The white, opaque thickenings of the inner pericardial surface so frequently found in adults are rare in children, but have been found at all ages, and where there is a deformity of the chest, as in certain cases of rhachitis, they have been especially noticed. Tuberculosis of the pericardium as a primary disease is even more rare in the child than in the adult. Tuberculosis secondary to tubercle of the pleura may occur, especially when the left pleura is affected. The younger the subject the less likely are there to be adhesions between the pericardium and the pleura, an important fact, to be taken into consideration later when I shall speak of the diagnosis of pericardial effusion in infancy.

**SYMPTOMS.**—Pericarditis may be acute or chronic, primary or secondary.

The subjective symptoms of acute pericarditis in infancy are very indefinite, and throughout childhood this latency of the early symptoms is so marked and occurs so frequently that it may be said to be characteristic of the symptomatology of pericarditis in early life. It is so difficult to locate pain when it occurs in the infant, and a tumultuous action of the heart with general circulatory disturbance is so commonly the result of a diseased condition outside of this central organ, that it is impossible to formulate a practical general symptomatology for the onset of the disease. When, however, the disease has progressed, dyspnœa and orthopnœa become marked. Large effusions appear to affect the functional activity of the heart more rapidly in children than in adults, and to occasion earlier the signs of disturbance of the circulation. Diminution in the amount of the urine in cases of pericardial effusion, with a corresponding increase in the urine as the effusion decreases, has been noticed in children. The usual physical signs supposed to be characteristic of pericarditis are often very misleading, and where a pericardial friction-sound is absent the determination of a case of pericarditis in a young child may present great difficulties. Owing to the flexible thorax of the child, there is a greater opportunity for the neighboring parts to yield before the pressure of an effusion, and we are more likely to have bulging of the intercostal spaces, and on inspection a visible alteration of the cardiac area, than in adults. Because of the small size of the child's thorax, the heart and pericardium are much nearer to the anterior surface of the thoracic cavity than they are in adults. This occurs both normally and in diseased conditions, especially where there is flattening, and thus levelling, of the chest. Under these latter conditions the heart and pericardium are brought in such close contact with the thoracic wall that on palpation you can feel the heart's impulse, and on auscultation the heart-sounds, in a much more advanced stage of a pericardial effusion than would be possible in the adult with a proportionately large amount of fluid. It has also been noticed in early life that on auscultation the sounds in pericarditis and endocarditis simulate each other quite closely. Percus-



sion is the most important physical sign, when the initial friction-sound has escaped detection, both for determining whether an effusion is present and as a guide to the prognosis and treatment. In effusions of exactly the same amount the area of dulness may differ, owing to the difference in the elasticity of the lungs and to the presence or absence of adhesions. The greater the elasticity of the lungs and the fewer the adhesions the more regular will be the outline of absolute dulness and the greater its significance as compared with that of the relative dulness; while the reverse of this proposition is true of the relative dulness. By *absolute dulness* I mean *entire absence* of resonance. By *relative dulness* I mean *diminished* resonance. The absolute dulness is determined by the retraction of the borders of the lungs, which withdraw from the chest-walls as the effusion gradually distends the pericardium. Relative dulness is due to the distended pericardium, and this to a greater or less degree compresses the lungs, which may be held in position by adhesions. The relative dulness, therefore, with its necessarily irregular outlines, varying according as adhesions are present or not, is most useful in studying complicated cases, while the absolute dulness should be relied upon in determining the outlines of the typical uncomplicated cases. The older the individual the more likely are adhesions and pulmonary diseases to be present. These will either prevent the retraction or alter the elasticity of the lung. The infant, being less likely to have had previous lesions of the lung and pericardium, gives us the best opportunity for studying the outlines of a pericardial effusion, and the area of absolute dulness is the most valuable physical sign of effusion in infants and in young children. It is evident, therefore, that we must acquire a precise knowledge of the uncomplicated cases before we shall be prepared to diagnosticate those in which adhesions are present or which are complicated by pulmonary disease. There is a great probability that many of the clinical observations on pericardial effusions made on adults by various competent clinical observers have been rendered of little practical value by the presence of adhesions, as has been proved by the difficulty in making a diagnosis by rules deduced from these observations. The number of clinical observations on infants corroborated by post-mortem examinations is not yet large enough to provide us with sufficient data from which to make precise deductions, but the experiments on which are based the diagrams of pericardial effusion which I am about to show you were made on sixteen infants, in none of whom did adhesions exist. In all of these presumably typical cases absolute dulness was found to the right of the sternum. An instance of how the area of dulness varies in complicated cases was given by a case in which, although the pericardium was much distended with fluid, it failed to show dulness to the right of the sternum, and the autopsy revealed adhesions binding the lung tightly to the right edge of the sternum. In this case the effusion was behind the lung, and this permitted resonance to be obtained over an area where in an uncomplicated case with the same amount of effusion there would have been dulness.

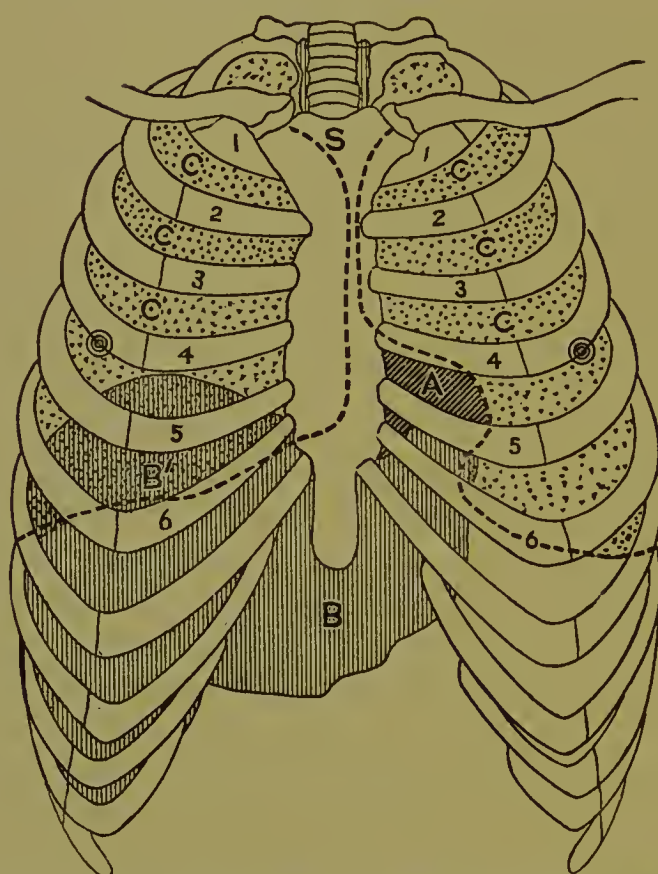
In addition to the difficulties in making a differential diagnosis arising from interference with the contractility of the lungs, such complications as pneumonia of the right lung, especially its middle lobe, pleuritic effusion on the right side, an enlarged liver, and an enlarged heart must be considered. Where this pneumonia, or pleurisy, or hepatic enlargement is present, an effusion into the pericardium cannot be diagnosticated by means of percussion, but these diseases can usually be readily determined by their especial symptoms. The differential diagnosis, on the contrary, from an enlarged heart, especially a dilated heart where the murmur may be absent, can often be made only by means of percussion.

Experiments with artificial effusions on the cadavers of healthy individuals should, therefore, first be made, and later further investigations be carried out, when possible, on individuals in whom the various conditions which interfere with the typical percussion outlines of a typical case are present. It is doubtful if these latter investigations will be carried out for many years, owing to the apparently insurmountable difficulties of producing these different abnormal conditions artificially. We can, however, learn much from the uncomplicated cases. Various methods of introducing fluids into the pericardium have been tried, and have failed to give satisfactory results. Although by dividing the sternum in the median line the pericardium can be entered without perforating the pleural cavity, yet when this method is employed the results of percussion are rendered void, since under these conditions air enters not only the anterior mediastinum but also the pericardium. The method which I finally devised and found to be most satisfactory in its mechanism was as follows. The infant was placed in the position of orthopnea; that is, the trunk was bent upon the lower limbs at an angle of about  $120^{\circ}$ . Tracheotomy was performed, and a clamped rubber tube was attached to the glass tracheal tube. The lungs were then inflated through this tube until on careful percussion the absolute area of cardiac dulness corresponded to that of a normal expiration. Under these conditions the area of absolute dulness, as you see in this diagram (Diagram 14, page 1050), begins at the junction of the upper border of the fourth left costal cartilage, and extends downward and outward to the left in a curved line, with the convexity outward and keeping 2 or 3 cm. ( $\frac{3}{4}$  or  $1\frac{1}{8}$  inches) within the nipple, until it joins the dulness of the left lobe of the liver. From the same starting-point at the fourth cartilage it extends downward in the left parasternal line, or rather within that line, towards the middle of the sternum, until it reaches the liver. The absolute dulness, therefore, is determined not by the shape of the heart itself, but by the marginal lines of the lungs, varying according to their expansion or retraction. This is a point which it is well to understand,—namely, that the pericardium itself, whether it is distended with fluid or not, does not by its own shape, as has been delineated so often in the plates illustrating pericardial effusions, aid us materially in determining the shape of the area of absolute dulness in a pericardial effusion. This area is marked by the retracting or rather



displaced borders of the lungs. After the inflation was accomplished the tracheal tube was clamped so as to retain the lungs in position. An incision was then made in the median line of the abdomen, from the pubes up to within 2 cm. ( $\frac{3}{4}$  inch) of the ensiform cartilage. The liver and stomach were gently drawn away from the diaphragm, and on palpation of the central tendon of the diaphragm four centimetres to the left of the median line

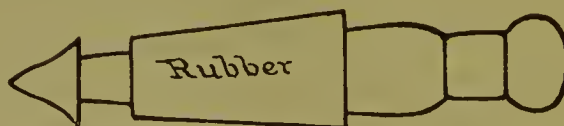
DIAGRAM 14.  
NORMAL THORAX.



■ A, physiological area of percussion-flatness of the heart on expiration; ▨ B, liver; ▨ B', that portion of the liver which is covered by the right lung; ▨ C, lung; S, sternum; ©, nipple; 1, 2, 3, 4, 5, 6, ribs; --- (broken line), border of lung.

the heart was felt. This point of the diaphragm was then carefully drawn down away from the heart, and a dagger-pointed trocar pushed through the diaphragm into the pericardial sac, which is adherent to the diaphragm at this point. Here is the trocar (Fig. 147) which, after many failures with other instruments, I finally devised, and have found to be satisfactory.

FIG. 147.

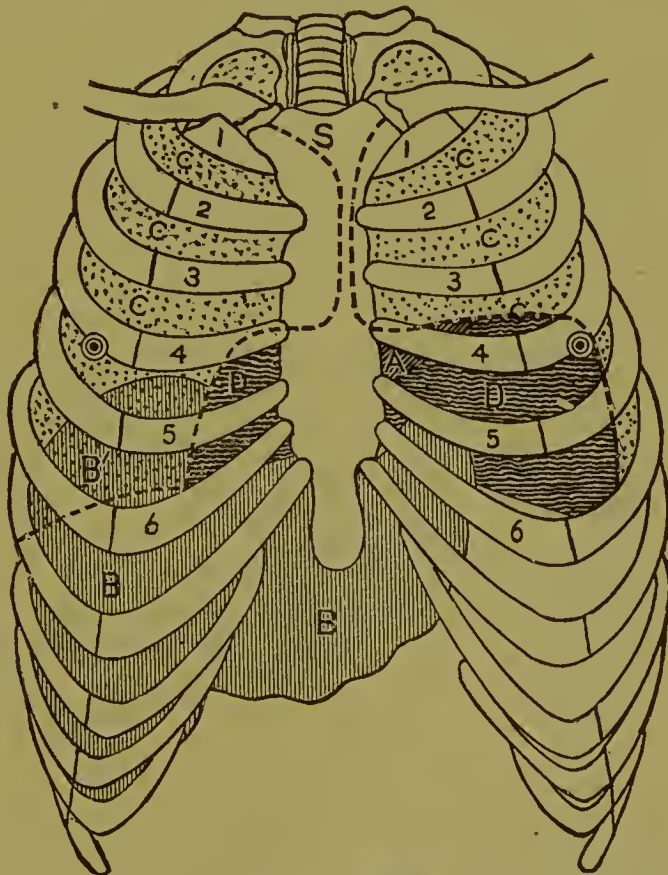


Artificial pericardial effusion trocar (full size).

The trocar is made of brass, with a conical point and a rounded shoulder forming the base of the cone, so that although it easily enters the pericar-

dium it is difficult to withdraw it, the point acting like the barb of a fish-hook. A short piece of rubber tubing fitted tightly to the neck of the trocar can, as soon as the point and shoulder have entered the pericardium, be pushed up tightly against the under side of the diaphragm, thus holding the trocar in position, and the diaphragm, being firmly compressed between the shoulder and the rubber tube, prevents the entrance of air. The trocar is connected by means of a piece of rubber tubing, which is also provided with a clamp, to a simple wash-bottle graduated for cubic centimetres and containing melted cacao butter. Before introducing the trocar the cacao butter is allowed to fill the tubing and the trocar so as to displace the air. As soon as the trocar has entered the pericardium the tracheal tube is unclamped, in order that the lungs may be free to retract before the fluid. When sufficient fluid, as indicated by the graduated bottle, has entered the pericardium, the cacao-butter tube and the tracheal tube are again clamped, the thorax is carefully percussed, and the line of absolute dulness is marked in ink. After twenty-four hours the sternum is removed from above downward, remaining attached below, and we find the lungs in position surround-

DIAGRAM 15.



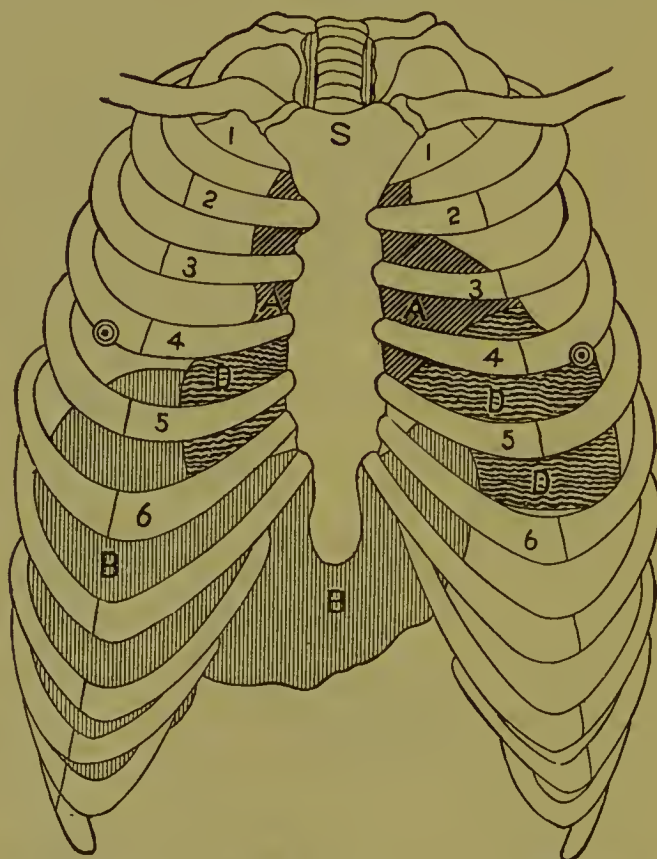
Small amount of liquid introduced into sac (Rotch). ■ A, the portion of the area of absolute dulness which is still caused by the physiological dulness of the heart; ▨ B, liver; ▩ B', that portion of the liver which is covered by the right lung; ☼ C, lung; ▤ D, effusion; A + D, area of absolute dulness found when the effusion is small; S, sternum; ⊙, nipple; 1, 2, 3, 4, 5, 6, ribs; --- (broken line), border of lung.

ing the hardened fluid. I have represented in this diagram (Diagram 15) the relations of the lung and the pericardium where a small amount of fluid has been introduced.



By replacing the sternum and verifying by means of needles penetrating the lines marked in ink, we can determine accurately the shape of the area of absolute dulness with this amount of effusion, which represents the results obtained when from 70 to 80 c.c. ( $2\frac{1}{3}$  to  $2\frac{2}{3}$  ounces) of fluid were introduced into the pericardium of an adult. There is a slight increase in the vertical as well as in the transverse area of dulness. The curved line which bounds the area of dulness starts at the sixth rib, to the right of the sternum, passes upward to the junction of the fourth cartilage with the sternum, impinges on the lower part of the third left interspace, and then descends just outside of the mammary line to the sixth rib, to pass inward until it meets the dulness of the left lobe of the liver. This line forms, as you see, an irregular semicircle, with a shorter radius to the right of the sternum and a longer one to the left. It is important to understand what causes this area of absolute dulness. This you can best do by referring to this next diagram (Diagram 16), where with the same amount of

DIAGRAM 16.



The lungs have been removed (Rotch). **A**, portion of the normal heart enclosed in the pericardium; **B**, liver; **D**, effusion as it appeared in the sac, the cacao butter being in small amount, and the lungs having been removed after the butter had hardened; **S**, sternum; **⊙**, nipple: 1, 2, 3, 4, 5, 6, ribs.

effusion the lungs have been removed, leaving the heart and the distended pericardium exposed to view.

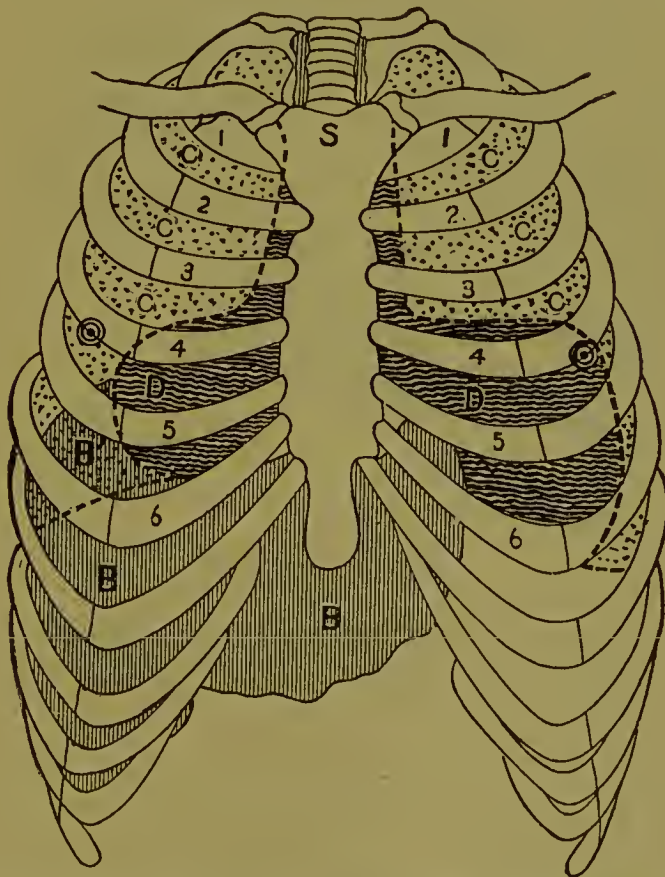
It will be seen on comparing the diagrams that a small section of the dull area, corresponding to the junction of the fourth and fifth ribs with the left side of the sternum, is formed by the heart itself, which is free

from effusion at this point, while the rest of the dulness is produced by the effusion. On examining also the hardened cacao-butter cast of this effusion, it was found that the layer of fluid was very thin all over the upper portion of the effusion in the region of the fourth rib and fourth interspace, while the mass of the effusion, as shown by the greatest thickness of the cacao butter, was, as would be expected from the laws of gravity and the shape of the pericardium, in the lower part of the sac on each side of the sternum in the fifth interspaces, the cast riding the arched diaphragm like a saddle, and the larger part of the mass being on the left side. These points should be carefully noted, as they are significant for diagnosis and treatment.

The same result as to the area of dulness was obtained with a proportionately small amount of fluid in an infant about two weeks old; and of sixteen injections, of infants of various ages, the areas of dulness were identical in all, and in all the lungs were normal and there were no pulmonary or other adhesions.

This next diagram (Diagram 17) represents the position assumed by the

DIAGRAM 17.



A large amount of liquid has been introduced into the sac (Roth). ■■■ B, liver; ■■■ B', that portion of the liver which is covered by the right lung; ■■■ C, lung; ■■■ D, the area of absolute dulness caused by a large effusion; S, sternum; ⊙, nipple; 1, 2, 3, 4, 5, 6, ribs; --- (broken line), border of lung.

margins of the lungs and the resulting area of absolute dulness where the pericardium was distended with a large amount of fluid so as to cover the entire heart.

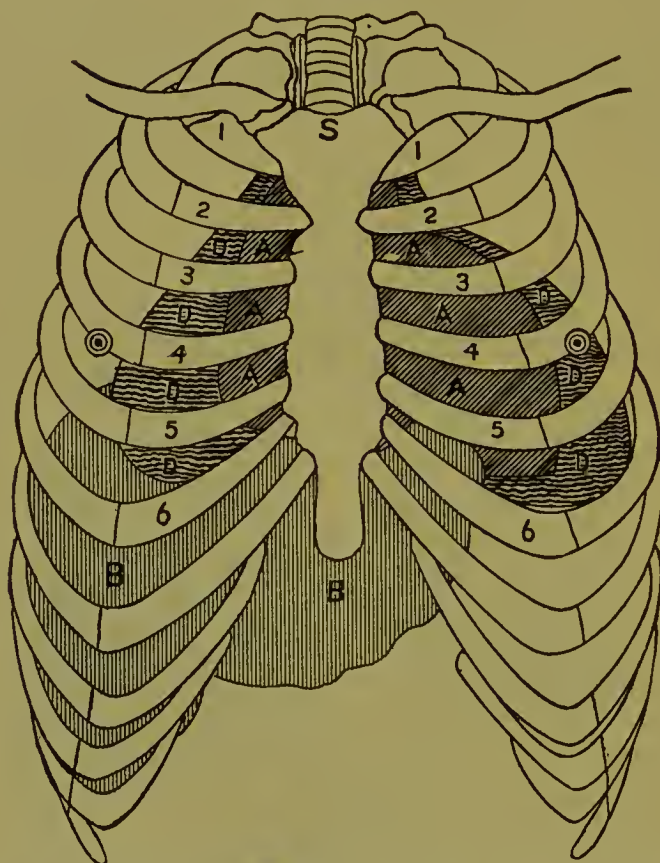
Here the transverse area of dulness produced by the much distended



pericardium has increased so that it extends farther to the right of the sternum in the fourth and fifth interspaces, and then, rising to the third interspace, it occupies a small area on either side of the sternum under the third, second, and first ribs and the second and first interspaces, the upper lobes of the lungs having retracted from beneath the sternum. As the effusion increases the lungs retract still more, and the upper curved lines of the effusion on either side of the sternum present areas with still greater diameters.

This next diagram (Diagram 18) represents this same large effusion with the lungs removed, and also shows the relations of the heart and great blood-vessels to the ribs and sternum before the pericardium has been distended with fluid.

DIAGRAM 18.



The lungs have been removed (Rotch). ■ A, normal shape of the heart in its pericardium; ▨ B, liver; ▩ D, effusion; A + D, the shape which the pericardium assumed in a case where considerable fluid had been introduced into the sac; S, sternum; ⊙, nipple; 1, 2, 3, 4, 5, 6, ribs.

As ordinarily seen on the injected subject, the heart would of course not appear as in the diagram, as it really was suspended in the pericardial sac with the effusion surrounding it and causing the entire area of dulness represented by A and D.

The fact that on opening the abdomen the diaphragm remains arched, and that the lung, by means of the tracheal clamp, retains its position and does not collapse, warrants us in assuming that we can fairly judge of the position of the fluid during life by this method of investigation, especially as the contractility and distensibility of the lung appeared to be perfectly retained after death, except in very cold weather, when it was found neces-

sary to warm the cadaver. It might have been objected to these experiments that the fluid was introduced at the bottom of the pericardial sac, while during life it might originate at the base of the heart. The fluid was, therefore, in several cases introduced where the pericardium is reflected over the great vessels; but even when it was in very small amount and insufficient to cause any increase of dulness, it immediately ran down the side of the heart to the bottom of the pericardium. Even when it was mechanically retained at the base of the heart by inverting the cadaver, the resulting cast had its broadest part towards the diaphragm.

DIAGNOSIS.—From what I have told you regarding the latency of the general symptoms of pericarditis in childhood and the difficulty of interpreting the local symptoms, it will be readily understood how important it is to recognize any especial symptoms which may characterize the disease. Instances have been reported where a distended pericardium was mistaken by experienced diagnosticians for an effusion into the left pleura.

The condition, however, which most closely simulates a pericardial effusion, both in its general symptoms and in its physical signs, is a dilated heart.

The most distinctive of all the physical signs of pericarditis is the friction-sound, when it is present. When, however, an effusion has taken place, the friction-sound may not be heard. This absence of a friction-sound is especially frequent in young children. The heart's impulse may be clearly perceptible, even when a considerable effusion is present, owing to the thin layer of fluid which covers the heart in the area between the left nipple and the sternum. We are therefore forced by the similarity which at times exists between the general symptoms, in the inspection, palpation, and auscultation of a dilated heart and of a pericardial effusion, to depend upon percussion in making a differential diagnosis. In order, however, to make a differential diagnosis between this area of percussion and that produced by an enlarged heart, it will be necessary to consider the possible area of dulness which may be produced by an enlarged heart, and, by comparing this area with that which I have shown to exist in a pericardial effusion, to determine the differential diagnosis between the two diseases.

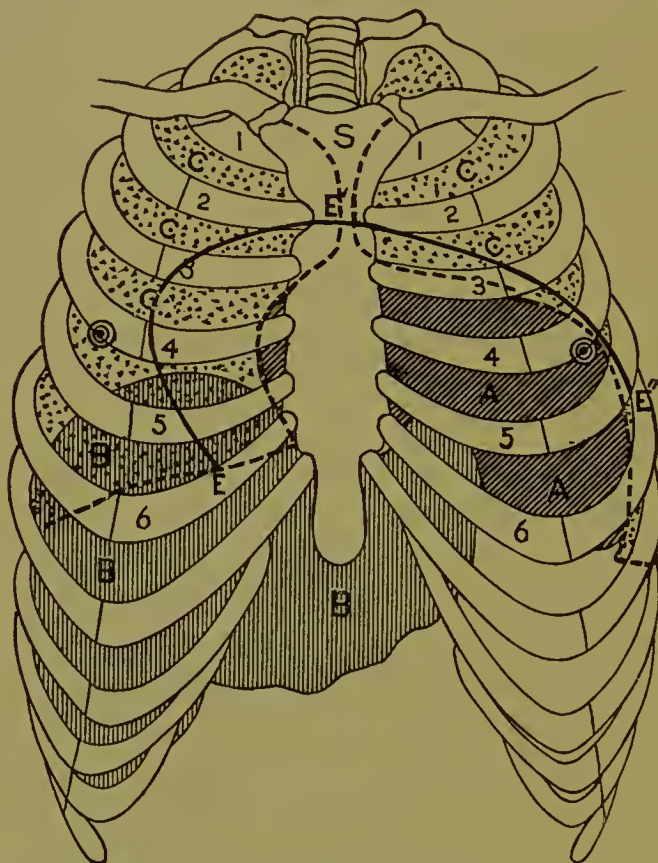
According to careful observations which have been made by competent observers on the area of dulness which can be produced by an enlarged heart, whether by hypertrophy or by dilatation, ventricular or auricular, although the relative dulness may extend to the right of the sternum from the second to the sixth rib, and in adults possibly to the distance of 3 or 4 cm. ( $1\frac{1}{8}$  or  $1\frac{1}{2}$  inches) on a level with the fourth rib, yet it is rare to find this dulness invading the fifth right interspace more than 2 or 3 cm. ( $\frac{3}{4}$  or  $1\frac{1}{8}$  inches), and still more rare for the absolute dulness to be found in the fifth interspace at all, and even in the fourth interspace for more than 1.5 or 2 cm. ( $\frac{1}{2}$  or  $\frac{3}{4}$  inch).

This diagram (Diagram 19, page 1056) represents the combined views of authorities on the dulness of an enlarged heart, and will be useful to refer to when we are considering the question of paracentesis. I have myself frequently verified these percussion-outlines, and in my experience it is exceed-



ingly rare, even in extreme cardiac enlargement, to find the relative dulness E E' E'' of as great an extent as is represented in this diagram. The absolute dulness as represented in the diagram I have met with in most cases of enlarged heart where the enlargement is great and the sternal region is involved.

DIAGRAM 19.



Enlarged heart (Rotch). A, area of absolute dulness caused by an enlarged heart; B, liver; B', that portion of the liver which is covered by the right lung; C, lung; E E' E'', the line marking the area of relative dulness of the enlarged heart; S, sternum; , nipple; 1, 2, 3, 4, 5, 6, ribs; --- (broken line), border of lung.

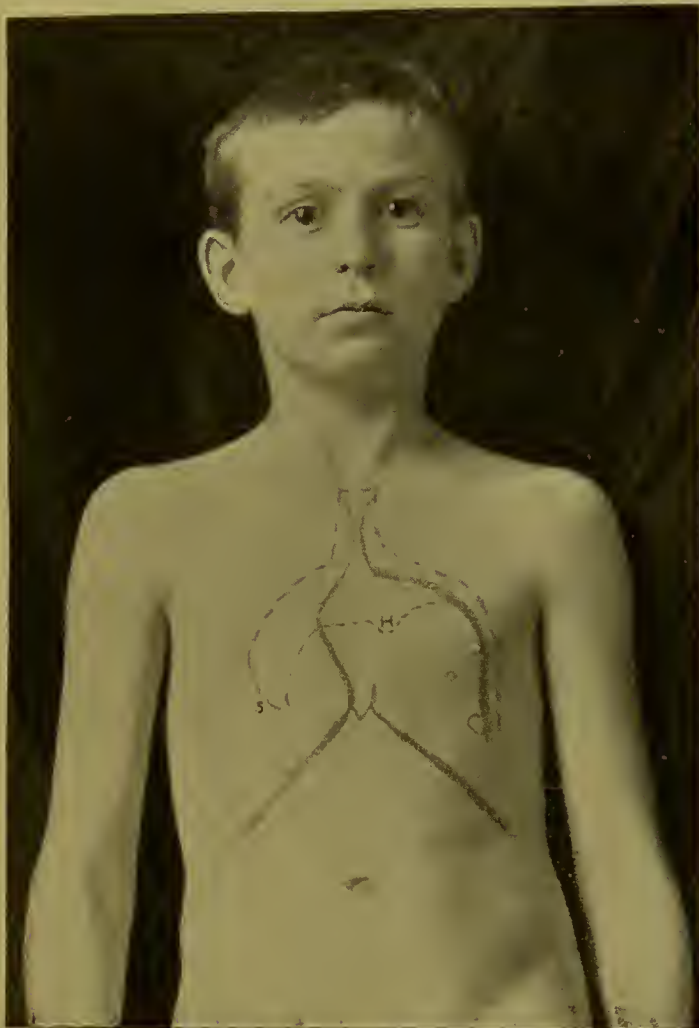
On referring to Diagram 17, we find that the dulness which occurs in a pericardial effusion may correspond to that of an enlarged heart through its whole area, but that the dulness of the effusion is also found in an additional area corresponding to a part of the fifth rib and fifth interspace. Absolute dulness, therefore, in the fifth right interspace 3 or 4 em. ( $1\frac{1}{8}$  or  $1\frac{1}{2}$  inches) from the right parasternal line in cases of pericarditis uncomplicated by pleural or pericardial adhesions becomes a valuable means of differential diagnosis from an enlarged heart.

I have found in my experiments on the adult pericardium that the absolute dulness could be detected in the fifth right interspace when from 70 to 80 c.c. of fluid had entered the pericardium.

In order to illustrate to you the difference between the area of dulness produced by an enlarged heart and that produced by a pericardial effusion, I have marked on this boy (Case 508, page 1057), eleven and a half years old, the boundaries of the area of absolute dulness in an enlarged heart, in a small pericardial effusion, and in a large pericardial effusion.

I have indicated the top of the sternum, the boundaries of the enlarged heart, the ensiform cartilage, and the lower border of the ribs by plain black lines, the boundaries of the small effusion by a broken line, and the area of the large effusion by a larger broken line. The figure 5 marks the

CASE 508.



Areas of absolute dulness in enlarged heart, and in distended pericardium. 5, fifth right interspace; H, heart.

fifth right interspace; the letter H marks that portion of the heart which has been left uncovered by the small effusion. The small black circle represents the normal position of the apex of the heart, the larger circle the apex of the enlarged heart. You will also notice how the enlarged heart extends beyond the right edge of the sternum at about the fourth rib and fourth interspace, and then returns beneath the lower part of the sternum within or very little outside of the right parasternal line. The outline of the small effusion, as well as that of the large effusion, is, as you see, to the right of the sternum as low as the sixth rib.

The following cases (Table 111, page 1058), taken from a number which have come under my care, illustrate the difficulty of making a differential diagnosis between cardiac and pericardial disease where, as at times happens, we fail to find a friction-sound or murmurs:



TABLE 111.

*Differential Diagnosis between Dilated Heart and Pericardial Effusion.*

CASE I. Endocarditis; Dilated Heart.	CASE II. Pericarditis; Effusion.	CASE III. Endocarditis; Enlarged Heart; Pericardial Effusion.
Girl, eleven years.	Boy, six years.	Girl, eight years. August 3, 1887.
Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.	Attack followed acute articular rheumatism.
Orthopnoea; præcordial pain.	Orthopnoea; præcordial pain.	Orthopnoea; præcordial pain.
Heart's impulse feeble, but perceptible a little to left and below left nipple, fifth interspace.	Heart's impulse feeble, but perceptible a little to left and below left nipple, fifth interspace.	Heart's impulse feeble, but perceptible all over cardiac area, with apex-beat a little below and to left of left nipple, fifth interspace.
Vertical absolute dulness not increased.	Vertical absolute dulness not increased.	Vertical absolute dulness not increased.
Absolute dulness under the sternum and to left of sternum; identical with Cases II. and III.	Absolute dulness under the sternum and to left of sternum; identical with Cases I. and III.	Absolute dulness under the sternum and to left of sternum; identical with Cases I. and II.
Absolute dulness did not extend to right of sternum.	Absolute dulness in fifth right interspace two or three centimetres from edge of sternum.	Absolute dulness in fifth right interspace three or four centimetres from edge of sternum.
Systolic murmur at apex.	Pericardial friction-rub at base.	Soft systolic murmur at apex, transmitted to axilla. Pericardial friction-rub at base.
Recovery.	Recovery.	<p>August 6: Less dulness in fifth right interspace; apex murmur much louder and harsh.</p> <p>August 11: Dulness only to right edge of sternum.</p> <p>August 18: Dulness only to middle of sternum; friction-rub ceased.</p> <p>December 1, 1887: Physical examination the same as on August 18, showing enlarged heart and mitral systolic murmur.</p>

You will observe that the symptomatology, both general and local, of these cases was, with the exception of the friction-sounds, murmurs, and percussion, identical, and that where an effusion was present dulness was

found in the fifth right interspace, while where it was absent dulness was not found. These typical cases with friction-sounds and murmurs were simply chosen in order that there should be no doubt as to the disease with which I was dealing when testing the value of percussion as a means for differential diagnosis.

I have referred to pericarditis with its accompanying effusion as being likely to occur in the later stages of scarlet fever. According to Steffen, when dilatation of the heart occurs in the later stages of scarlet fever, in cases where from the age of the child, three to eight years, the physiological hypertrophy of the heart is present, the tendency to enlargement is still further promoted by the increased blood-pressure from the diseased kidney, and a differential diagnosis between a pericardial effusion and an enlarged heart thus becomes necessary.

In connection with pericarditis we should consider the possibility of both complete and partial obliteration of the pericardial cavity occurring in children. Where severe cardiac symptoms are present and no valvular murmurs are heard, we should in young children think of degeneration of the heart-muscle itself or of pericardial adhesions. When, again, the absolute area of dulness remains unchanged and there are well-marked systolic retractions, the presence of pericardial adhesions is highly probable.

While in older children and in adults pericarditis is manifested by weakness of the apex-beat, the latter sometimes being imperceptible, and by a friction-sound, in very young children these symptoms are often absent, because the exudation is moderately thick and may not be abundant enough to cause friction-sounds or to mask the apex-beat.

*Chronic pericarditis* may occur in infancy and in childhood as in adult life, and is the result of acute inflammatory processes which have resulted in adhesions. It is often very latent, as is shown by autopsies.

PROGNOSIS.—In early infancy diffuse pericarditis is a very dangerous disease, and usually soon ends fatally. In later childhood its course and results are much the same as in adults, and in the acute form the disease has a tendency to recovery. Among the unfavorable complications of the disease which render the diagnosis especially serious may be mentioned adhesions of the two layers of the pericardium, which may paralyze the cardiac muscles and from the resulting stasis of blood may lead to extensive dropsy. The principal symptoms of this form of cardiac paralysis are a small and frequent pulse, subnormal temperature, oedema of the cheeks, lids, and lower extremities, and the presence of a small quantity of albumin in the urine.

TREATMENT.—The treatment of pericarditis in infancy and in early childhood does not differ materially from that in later life, and depends upon the various causes which I have referred to in speaking of the etiology of the disease. The tendency to heart-failure, however, which is so pronounced in the child, should be guarded against. Early in the disease absolute physical and mental rest should be enforced. In the acute



stage of the disease, before an effusion of any extent has formed, cold can be applied to the cardiac region by means of coils of tubing containing ice-water. An important part of the treatment is the judicious administration of digitalis to aid the heart in the crippled condition in which it is usually left after the early days of the disease. Stimulants should be freely used when there is any indication of heart-failure.

The most important part of the treatment when an effusion of any extent has occurred is paracentesis of the pericardium, which should unhesitatingly be performed, no matter what the cause of the disease may be, when life is in danger from undue distention of the pericardial sac. The pericardium has usually been aspirated to the left of the sternum. The possibility of wounding the heart when the aspiration is made to the left of the sternum should be considered, and, if possible, avoided. An important point both in the diagnosis and in the treatment should be here spoken of. It has been held by certain authorities that the heart's apex is found in effusions to be tilted upward and inward towards the sternal end of the fourth left interspace,—that is, floated up by the effusion. Direct proof of this is wanting, and I believe, from my investigations on this subject, that it is an erroneous view. It would seemingly be impossible for the heart not to sink rather than to be floated up, unless the specific gravity of the effusion was greater than 1050, as I have shown by experiment. It is highly improbable that the specific gravity would be greater than 1050 in an ordinary pericardial effusion, for the specific gravity of a purely purulent fluid is only about 1032. How, then, can we explain the clinical phenomenon of the heart-beat in the region of the fourth left interspace, where it is so frequently found in cases of pericardial effusion? Referring to Diagrams 15 and 16, and to Case 508 (pages 1051, 1052, 1057), it seems plausible to account for this pulsation by the tumultuous action of that portion of the right ventricle which is seen to be free from the effusion in the fourth left interspace when an effusion of any extent is present.

On examining the cacao-butter casts it is also found that this portion of the heart is in the larger effusion, as I have already described to you, covered by a very thin layer of fluid, through which the impulse of the heart can easily be felt and seen. This fact is of especial significance when we consider that both Ludwig and Bowditch have observed that the impulse of the heart as seen normally in the fifth left interspace need not be caused by the heart's apex, but may be caused by a portion of the heart above the apex striking against the thoracic wall. We should also consider that the impulse of the heart in children is often chiefly in the fourth interspace. In Case III. described in this table (Table 111, page 1058) it is recorded that the impulse was found through the whole cardiac area, but that it was still pronounced in the fifth interspace. Now, if in this case there had been a larger effusion, the apex and the lower segment of the right ventricle being surrounded by a mass of fluid, the impulse would have been lost in the fifth interspace, while in the fourth interspace, where the ventricle is covered by

only a thin layer of overlying fluid, the impulse would have continued to be both seen and felt, thus simulating an apex-beat. I believe that this is the explanation of what has been called misplaced apex-beats and floating upward of the heart in pericardial effusions.

From the above facts,—namely, that the heart, when an effusion is present, remains in its usual position, and does not, even when much enlarged, impinge on the fifth right interspace, and that the effusion, even when in so small an amount as 100 c.c., is found in the fifth right interspace,—is it not more rational to choose the fifth right interspace as the point for tapping, thus avoiding all question of injuring the heart? When we tap the pleura, we avoid the heart as much as possible: why not carry out the same rule in paracentesis of the pericardium? I have tapped the pericardium in the fifth right interspace a number of times on the cadaver, and have removed the fluid as easily as in the fifth left interspace.

The pericardium has been tapped during life in the fifth right interspace by Ebstein, of Göttingen, and Wilson, of Nashville.

As an illustration of how important it is to tap the pericardium when it is much distended with fluid and when symptoms of failing heart have arisen, I shall report to you a case which occurred a few days since in the wards.

A boy (Case 509), six years old, entered the hospital with a history of having had œdema of the face, hands, feet, and ankles for four weeks. There was no history of rheumatism, and the case was apparently one of acute primary endocarditis with mitral insufficiency. The cardiac area of dulness was increased, and extended from the middle of the sternum to 1.5 cm. ( $\frac{1}{2}$  inch) beyond the left mammary line, where the impulse of the heart could be felt. The child was kept quiet in bed, and after a few days the œdema lessened and he was very comfortable. While still under treatment, two weeks later, the temperature, which had been normal, rose to 39.1° C. (102.5° F.), the pulse was quickened and somewhat irregular, and the respirations were increased. A few days later a pericardial friction-sound was heard over the upper part of the sternum, and the temperature fell to 37.7° C. (100° F.). There was no change in the cardiac area of dulness, and no evidence of a pericardial effusion.

On the following day the cardiac sounds were found to be rather muffled; the child did not seem so well, and was unable to lie on his left side. Two days later the area of præcordial dulness extended farther to the right, and a little beyond the right parasternal line in the fifth right interspace. The attendants were directed to watch the child closely, and warning was given that the necessity for paracentesis of the pericardium might at any time arise. Early the following morning the child began to have marked dyspnoea and became very cyanotic. The house-officer found that the præcordial dulness had extended 2.7 cm. (1 inch) beyond the right edge of the sternum in the fifth interspace, and he therefore got the instruments ready for performing paracentesis. Suddenly the child's pulse became very weak and intermittent, the cyanosis increased very much, the dyspnoea became very marked, and, although stimulants were quickly given, the child suddenly gasped and fell back on its pillow dead. This occurred within three-quarters of an hour from the time when the first serious symptoms arose. The house-officer, Dr. Stickney, immediately introduced the aspirating needle in the fifth right interspace and withdrew some fluid from the pericardium. The child, however, did not revive.

This case of pericardial effusion, as well as the case of pleuritic effusion (Case 481, page 1011), should warn us that whenever a pleural or a pericardial effusion is present the child should be watched with the greatest care, and paracentesis should be performed as soon as any urgent symptoms arise.



Here is a little girl (Case 510), six and one-half years old. She has never had rheumatism, but she had an attack of measles when she was two years old, pertussis when she was two and a half years old, and parotitis when she was three and one-half years old. Four months ago she had an attack of chorea, of so mild a grade, however, that she has been able to go to school until entering the hospital. At that time, although she did not show any especial cardiac symptoms, an examination of the heart showed a latent and insidious endocarditis, represented by an increase of the cardiac area of dulness to the left of the mammary line, but not extending under the sternum, with a systolic murmur transmitted to the axillary line, but not heard in the back. Compensation soon became complete, and she recovered from the chorea.

Two days ago she was attacked with dyspnœa, rapid respirations, and cardiac pain.

#### CASE 510.



Chronic endocarditis. Mitral insufficiency. Pericarditis sicca. F, pericardial friction-sound; 5, fifth right interspace.

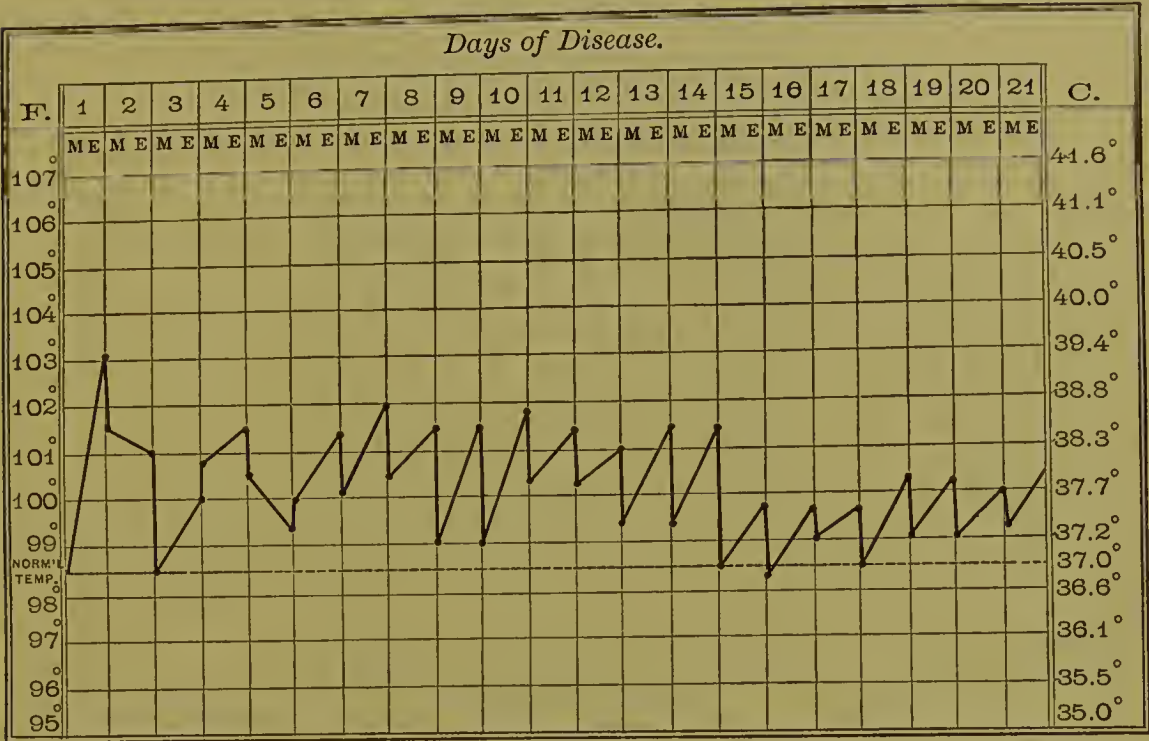
On examining her to-day the child seems very sick, and in addition to the area of cardiac dulness which I have marked in black, and which, as you see, shows the presence of a dilated heart, I find at the junction of the third rib with the sternum a marked præcordial friction-sound.

(Subsequent history.) The præcordial pain, discomfort, and heightened temperature lasted for a few days, and were in the beginning accompanied by orthopnœa and by the friction-sound becoming more intense. There was at no time, however, any evidence of an effusion in the pericardium, and one week afterwards the friction-sound became less distinct, disappearing three days later. The child, however, grew much weaker, and, although she was treated by complete rest in bed and with digitalis, strophanthus, and stimulants, the præcordial pain returned, and she gradually failed and died. The chart (Chart 46, page 1063) shows the temperature during the attack of pericarditis. The pulse varied from 130 to 150, and the respirations from 50 to 80.

The autopsy showed the pericardial sac to be obliterated everywhere by firm fibrinous adhesions. The heart was enlarged. Along the edge of the mitral valve were numerous small grayish-white vegetations. These were also present on the aortic valves and on the portion surrounding the tricuspid valve. The lungs were denser than normal, and were deeply injected and œdematous. The pleura on the inner surface of the right lower lobe was

adherent to the pericardium by fibrinous adhesions. The surface of the liver was covered with a thin layer of fibrin. The liver and kidneys were a little denser than normal, but were not noticeably congested.

CHART 46.



Pericarditis sicca.

Anatomical Diagnosis.

- Chronic adhesive pericarditis.
- Acute vegetative endocarditis.
- Acute fibrinous pleurisy.
- Acute fibrinous perihepatitis.
- Hypertrophy and dilatation of the heart.

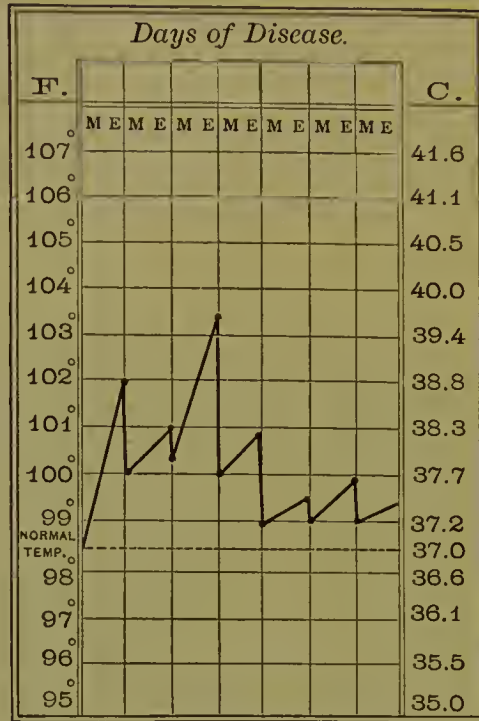
Here is a little girl (Case 511), eight years old, who during the first two years of her life had scarlet fever, varicella, and pertussis. When she was two years old she had an attack of measles, and when she was seven years old an attack of chorea. During the last year she has been fairly well until two weeks ago, when it was noticed that her feet began to swell, she complained of pain in her limbs, and occasionally of headache, she lost in weight, and lately has had orthopnoea with frequent paroxysms of dyspnoea. She has also at times complained of pain in her left chest. Her extremities are apt to be cold. For the past two days she has had a short, dry cough. A physical examination shows the impulse of the heart to be feeble, but it can be felt all over the cardiac area. There is an area of præcordial dulness extending to the right of the sternum almost to the right mammary line, as low as and involving the fifth interspace and as high as the third interspace and to the left a little beyond the left mammary line to the sixth rib. There is a systolic murmur at the apex, which is transmitted to the axilla. The pulmonic second sound is accentuated. There is a præcordial friction-sound heard at the upper part of the sternum. The history of the case and the area of præcordial dulness show us that it is a case of pericarditis with effusion. There may also be some endocarditis, evidence of which is given by the mitral systolic murmur. The child is being treated by rest in bed and by digitalis.

(Subsequent history.) Two weeks later the friction-sound disappeared, and the præcordial dulness grew less, so that it extended only to the middle of the sternum. In the next two weeks the dulness beneath the sternum disappeared, and the mitral murmur lessened, but could still be heard 2 cm. ( $\frac{3}{4}$  inch) outside of the mammary line.



This chart (Chart 47) shows the irregular temperature during seven days of the pericardial effusion.

CHART 47.



Pericarditis with effusion.

# DIVISION XVIII.

## UNCLASSIFIED DISEASES.

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### LECTURE LIII.

RHACHITIS.—SCORBUTUS.—RHEUMATISM.—PURPURA.—DIABETES.—TUBERCULOSIS.—  
EPIDEMIC INFLUENZA.—DISEASES OF THE THYROID GLAND.—DISEASES OF THE  
CERVICAL LYMPH GLANDS.—PAROTITIS.—DISEASES OF THE EAR.

I SHALL now speak of a number of diseases which are not readily classified under the divisions that I have found most useful for teaching, and which will therefore have to be spoken of separately.

**RHACHITIS.**—Rhachitis is a disease of infancy, rarely of early childhood, and is closely associated with impaired nutrition. It shows itself mostly in alterations of the growing bones. Its most marked symptoms are met with between the sixth month and the second year, but it can occur at all ages, and may be congenital.

**ETIOLOGY.**—Although the cause of rhachitis is not yet clearly understood, it is evidently closely connected with interference with the nutrition by improper food and lack of suitable hygienic surroundings. Although it most frequently exists after the first six months of life, yet probably many cases occur earlier, but are so mild in form that the rhachitic lesions do not become sufficiently marked for recognition until the latter part of the first year. It is well known that rhachitis is much more common in its occurrence among all classes of life than was formerly supposed. As a congenital disease it is probably associated with lack of proper intra-uterine nourishment, corresponding to the rhachitis which is met with in cases of prolonged lactation. Like all diseases associated with impairment of nutrition, it is less likely to occur among breast-fed infants than among those who are deprived of their natural food. For the same reason it is more likely to develop in the latter part of the first year than in the early months, since in so many cases the breast-milk deteriorates in quality after the first six or seven months of lactation. The disease seems to occur where the food is not properly adapted to the especial age. Certain races, such as the lower classes in Italy and in England, are notably affected by rhachitis. The disease in a marked form is not common among native-born Americans.



**PATHOLOGY.**—Although there are lesions of the various organs which seem to be closely connected with rhachitis, such as enlargement of the spleen and of the liver, yet the bones show so markedly the most important changes that practically and clinically, in the present state of our knowledge concerning the disease, these changes in the bones constitute its pathology. We must remember, however, that the nutrition of all the tissues is profoundly affected, and that the equilibrium of the nervous system is very unstable.

According to Delafield and Prudden, the growth of the bones depends upon three conditions. They grow in length by the production of bone in the cartilage between the epiphysis and the diaphysis, and in thickness by the growth of bone from the inner layers of the periosteum. At the same time the medullary canal is enlarged in proportion to the growth of the bone by the disappearance of the inner layer of bone. In rhachitic children these three conditions are abnormally affected. The cartilaginous and subperiosteal cell growth which produces ossification goes on with increased

FIG. 148.



I. Normal bone : Z. P., zone of proliferation. II. Bone of a cretin : Z. P., zone of proliferation.  
III. Rhachitic bone : Z. P., zone of proliferation.

rapidity and in an irregular manner both between the epiphysis and the diaphysis and beneath the periosteum, while the actual ossification is markedly irregular or wanting. At the same time the dilatation of the medullary cavity goes on irregularly and often to an excessive degree. If we examine microscopically the region between the epiphysis and the diaphysis usually called the zone of proliferation, we find that the cartilaginous cells are not regularly arranged in rows around a definite zone in advance of the ring of ossification, as in normal tissue, but that there is an irregular heaping up

of cartilaginous cells, sometimes in rows, sometimes not, covering an ill-defined irregular area. This zone of proliferation also, instead of being narrow and sharply defined, is quite lacking in uniformity. Areas of calcification may be isolated in the region of the proliferating cartilaginous cells, or calcification may be altogether absent over considerable areas.

Here is a section of a normal bone (Fig. 148, I.) taken from an infant which shows the normal zone of proliferation (Z. P.) between the epiphysis and the diaphysis.

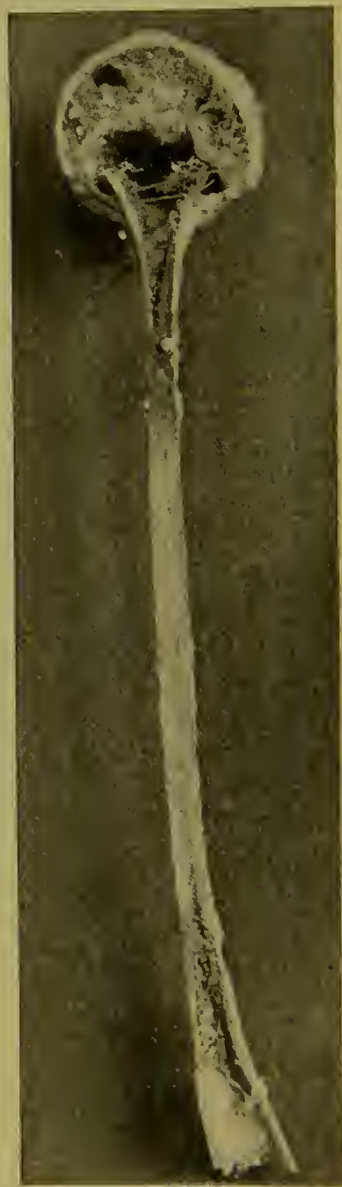
Here also is a section of a rhachitic bone (Fig. 148, III.) which shows the broad, irregular, and abnormal zone of proliferation (Z. P.) which I have just described.

Here is a section of another rhachitic bone (Fig. 149), which shows the great enlargement of cartilage at the epiphysis, with the irregular foci of calcification. The diaphysis of the bone shows periosteal thickening to such an extent that it encroaches on the medullary cavity, which, as you see, is much diminished.

An excessive proliferation of cells in the inner layers of the periosteum, the irregular calcification which occurs about them, and the absence of uniformity in the elaboration of the structure of the bone, produce an irregular, spongy bone-tissue instead of the compact lamellated tissue which is so necessary for the uniformity of the structure. The increased cell-growth between the epiphysis and the diaphysis produces the peculiar knobby swellings which are characteristic of rhachitis. At the same time the medullary cavity increases rapidly in size, and the inner layers of the bone become spongy. The medulla may be congested, and fat, if it has formed, may be absorbed, and a species of osteitis ensue. The result of these processes is that the bones do not possess solidity and cannot resist the strain of the muscles or outside pressure. After a time the rhachitic process may stop and the bones may assume a more normal character. The porous bone-tissue becomes compact, and even unnaturally dense, so that in later childhood the rhachitic bone is unusually hard, like ivory, a condition noticed by those who have to operate on these bones.

The swelling at the epiphyses disappears as the disease passes off. Many of the deformed bones may become of a normal shape, but in severe cases the deformity may continue through life, especially if there is a cessation of the growth of the bones in their long axes, so that the children are dwarfed.

FIG. 149.



Spindle-shaped rhachitic bone.



The first signs of rhachitic disease are always found in those parts of the bones which are in the most active stage of development. In the early days of extra-uterine life the skull undergoes the most marked changes. The cranium may be unusually large for the size of the face. The fontanelles and sutures are widely open. The bones may be soft, porous, and hyperæmic, while at their edges there may be rough bony projections beneath the periosteum. Sometimes, especially in the occipital bone, there are rounded defects in the bone filled only with a fibrous membrane. This constitutes what is called *craniotabes*. The head itself is usually large and square, in contradistinction to the hydrocephalic head which I showed you at an earlier lecture (Case 286, page 638), and which in consequence of the eversion of the parietal bones has a globular shape. In the rhachitic head the parietal bones are more vertical, thus giving the square appearance.

The forehead is sometimes very prominent, and the normal thickness of the bones is increased by means of a large amount of new periosteal soft growth between the periosteum and the bones, which produces this marked deformity of the forehead.

The forehead looks high and square, and the top of the head is usually depressed. This condition of the bones may be only temporary, but if there is much deposit under the periosteum it will sometimes remain, and

CASE 512.



Rhachitic head. Male, 3 years old.

where calcification takes place quickly the thickened areas of the bone will remain unabsorbed throughout life. These areas of thickening, however, are often absorbed.

The teeth in rhachitic children are late in developing, and the intervals between the appearance of the different groups are longer than normal. The lower jaw is apt to be short and its angle sharp and prominent.

This infant (Case 512) shows the square rhachitic head.

In striking contrast to the large square head is the narrowed and flattened thorax. There is usually a compression of the chest laterally and a protrusion of the sternum and lower ribs, due to a constriction following the line of the diaphragm. The costal cartilages are frequently enlarged at the junction with the ribs, and can be felt and often seen as a line of rounded prominences. These prominences are called the *rhachitic rosary*. This rosary, although most commonly occurring in the latter part of the first year, has been noticed by Jacobi at the age of two months, and it has also been met with in the early weeks of life. The sternum may be depressed, or with the costal cartilages it may be pushed forward, forming what is known as *pectus carinatum* (pigeon-breast).

FIG. 150.



Inner surface of sternum, with cartilages and portions of ribs attached, showing rhachitic rosary.

Here is a specimen (Fig. 150), taken from a rhaehitic child, of the sternum to which are attached the cartilages and portions of the ribs.

It shows on the inner surface a distinct rosary. During life this rosary could not be detected on the outer surface of the thorax.

This infant (Case 513, page 1070) shows very markedly a rhaehitic rosary, with depression of the lower part of the thorax, and enlarged epiphyses at the wrist.

I have described in previous lectures (pages 71, 1019, 1045) the various deformities of the sternum which arise in connection with a delay in ossification, and which may also occur in such defective ossification as takes place in rhaehitis. In addition to these anterior and lateral deformities of the thorax, kyphosis is quite frequently seen in cases of rhachitis at the junction of the lumbar spines when the children begin to stand erect and to walk. Lordosis may be present. Lateral curvature may also occur.



This child (Case 514) shows rhachitic kyphosis to a marked degree.

I shall not attempt to describe all the deformities which may arise in rhachitic bones. They are very numerous, and, although exceedingly inter-

CASE 513.



Rhachitic rosary and enlarged epiphysis of the wrist. Female, 25 months old.

esting and important, are in the province of the orthopædic surgeon rather than in that of the physician. A well-marked deformity in connection with the limbs is the enlargement of the epiphyses, which I have just described

CASE 514.



Rhachitic kyphosis. Female, 3 years old.

when showing you this bone (Fig. 149, page 1067). These enlargements are especially noticeable at the wrists and ankles. The legs are apt to be bowed. Knock-knee is also often a result of rhachitis.

This child (Case 515, page 1071) shows a number of rhachitic deformities.

She has a square head. The thorax is narrow and contracted. The sternum is prominent. The epiphyses of the ankles and wrists are much enlarged. The arms are bowed.

Slight lateral curvature is present. The abdomen is distended. She is bow-legged and knock-kneed, and has flat-foot.

Here is another child (Case 516) who shows markedly the rhachitic deformities of the wrists, the distended abdomen, the rosary, and the rhachitic head.

CASE 515.



Rhachitic deformities.

CASE 516.



Rhachitic deformities. Male, 2 years old. The enlarged epiphyses of the ribs are marked with black spots.

**CONGENITAL RHACHITIS.**—Although the occurrence of intra-uterine rhachitis has been disputed, yet there seems to be sufficient evidence of such a disease in new-born infants to warrant the statement that rhachitis may be met with in this early stage of existence: it is, however, a very rare affection. I have seen a case of congenital rhachitis in which the rhachitic process had run its course and the hardening of the bones had apparently been completed before the infant was born.

Another case of congenital rhachitis which has come under my observation was seen by me in consultation with Dr. Townsend (Case 517, page 1072). The parents were young and healthy, and there was no history of syphilis or rhachitis. The father was American, the mother Scotch. There was one other child, three years old, strong and well. The mother during her pregnancy was much worried, and her nourishment was both insufficient and poor. The infant, a male, was one month premature. The labor was easy. The infant weighed seven pounds and was 43.3 cm. (17 inches) in length.

I have here a photograph which was taken on the fourth day of the



infant's life. The head, as you see, was square in front, was much flattened behind, and measured 33.8 cm. ( $13\frac{1}{4}$  inches). The sutures were all widely open. The ossified portions of all the bones of the skull were small, particularly of the occipital bone, which presented a large area of craniotabes. In the widely-opened sagittal suture just back of the anterior

CASE 517.



Congenital rhachitis.

fontanelle was a large Wormian bone 2.7 cm. (1 inch) long. In the squamous and coronal sutures on the right side at least eight small Wormian bones could easily be felt, and on the left side eleven were counted. The thorax was 30 cm. ( $11\frac{3}{4}$  inches) in circumference, and was depressed laterally, the depression increasing with each inspiration, owing to an accompanying atelectasis in the lower portions of the lungs. There was considerable cyanosis. No cardiac murmur was detected. A rachitic rosary was present. The abdomen measured at the level of the umbilicus 28.7 cm. ( $11\frac{1}{4}$  inches). There was a large double inguinal hernia. The spleen could not be detected on examination. The liver could be felt below the edge of the ribs, but was apparently not enlarged. There were marked enlargement of all the epiphyses, curvature of all the long bones, and numerous fractures. The humeri showed a slight anterior curvature. The bones of each forearm were also bent anteriorly. The femora were curved outward and forward. The lower legs showed marked angular curvatures forward at the junction of the middle and lower thirds. The fractures were apparently of as recent origin as the birth, as some of them proceeded to unite very quickly. On the eighth day the fracture of the right tibia was quite firmly united; and only a slight crepitus could still be felt over the left tibia. The fracture of the left humerus was firmly united with a ring of callus. The right humerus at birth showed a callus about the middle of the shaft: this was evidently the repair of an intra-uterine fracture. The child died on the ninth day of its life.

**SYMPTOMS.**—The symptoms of rhachitis are those of a slowly developing constitutional disease. The early symptoms are those which may occur in a number of diseases, and are closely connected with disturbance of the gastro-enteric tract. The children, although they are often quite heavy, are anæmic, and their muscles are soft. The increase in weight depends more on the increase of fat, the normal relative proportion between fat and muscle being altered. Their appetite is capricious; they become fretful, and perspire at night, especially about their heads. They do not learn to walk so early as does the normally developed infant, and they soon show the later and more characteristic signs of rhachitis. I have already spoken of these signs when describing the pathology of rhachitis. As a rule, however, the picture of a rhachitic child is one with a square, prominent forehead, and with an anterior fontanelle remaining open after the age of eighteen months; dentition is delayed; the thorax is narrow and compressed laterally; the rhachitic rosary and enlargement of the epiphyses of the wrists and ankles are present, and the abdomen is distended. The bones of the extremities may be bowed, and the feet may be flat. In some cases there is considerable tenderness of the bones and muscles. The muscles are often so weakened by the depressing effects of the disease that the child has not sufficient strength to walk steadily. There are also a series of nervous phenomena connected with rhachitis which play a very prominent part in the disease. Convulsive attacks are more frequent in rhachitic children than in those whose nervous system is in equilibrium. The condition of laryngospasmus, which I have described to you in previous lectures (pages 747, 949), is at times a prominent feature in the symptoms of rhachitis. Rhachitic children are more liable to die than other children when attacked with such diseases as pneumonia or bronchitis. Attacks of the acute exanthemata are of serious import in rhachitic children, and these children are especially liable to the invasion of the bacillus tuberculosis.

**DIAGNOSIS.**—The diagnosis of rhachitis should be made from a number of diseases in which the general nutrition of the child is profoundly disturbed. When the disease is fully developed the diagnosis is not difficult. In its early stages, however, the manifestations of rhachitis may be so slight that the diagnosis must often be kept in abeyance. I have already spoken of the diagnosis of rhachitis from hereditary syphilis, and, as a rule, no difficulty arises. You must remember that syphilis and rhachitis have no direct connection with each other, but are both chronic constitutional diseases, and that it is possible to have both diseases occur in the same individual. I have described the syphilitic bone in a previous lecture (page 497). When there is enlargement of the long bones it is not limited to the epiphyses, as in rhachitis, but involves the ends of the diaphysis. It is often accompanied by a condition which closely simulates a callus, and there is a distinct tendency to fracture in syphilis rather than to the bending which is common in rhachitis. The notched teeth and the cranio-tabes may occur in both diseases, while the lesions of the mouth and



lips, which I have already fully described (page 494), are distinctive of syphilis.

The diagnosis from scorbutus I shall speak of presently (page 1077).

The heightened temperature and the acute tenderness and swelling of the joints in acute articular rheumatism are easily distinguished from the subacute or chronic course and the characteristic enlargement of the epiphyses in rhachitis.

Rhachitis, where it causes kyphosis of the spine, may simulate Pott's disease very closely. It occurs at the dorso-lumbar junction, which is a frequent seat of the deformity in Pott's disease. The spine is held rigidly in severe cases, just as in Pott's disease, and the deformity may be angular rather than the usual gradual curve. The coexistence of enlarged epiphyses and other rhachitic conditions makes it very probable that the affection is rhachitic; but both diseases may coexist.

In general, the age of the child, the absence of much pain, and the existence of other signs establish the diagnosis of rhachitis. It is, moreover, in children under two, much more common than Pott's disease. In doubtful cases the diagnosis can be made only after several examinations and a period of two or three weeks of recumbency, under which conditions the rhachitic spine becomes somewhat more flexible.

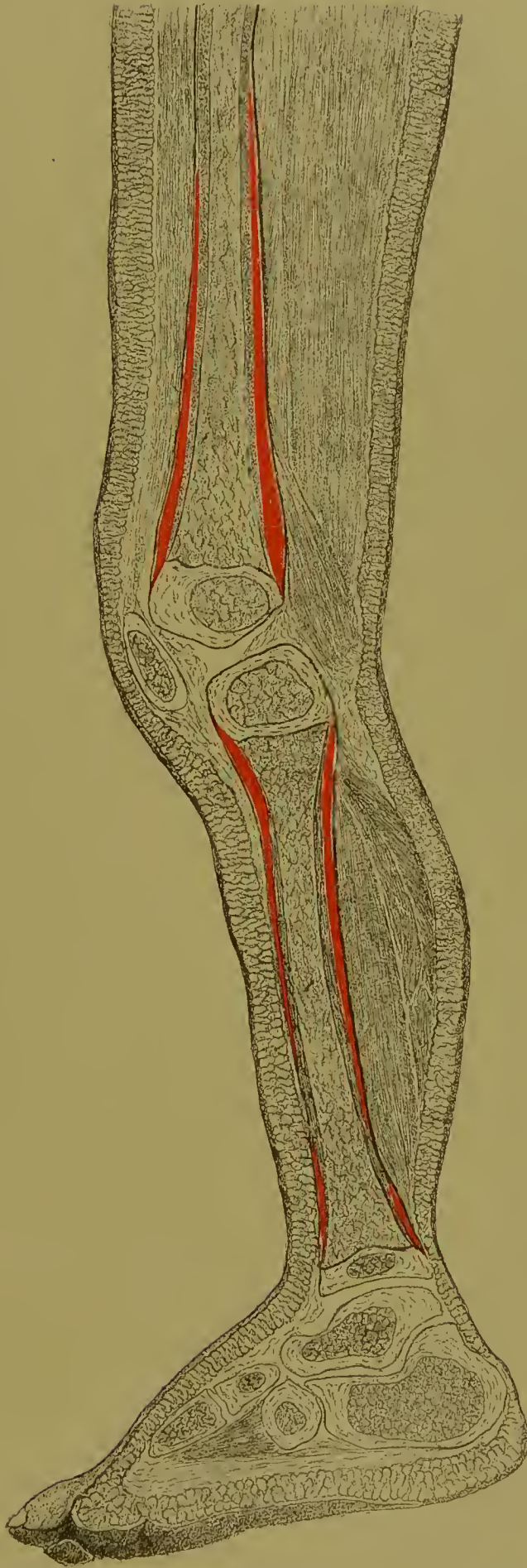
Cases of rhachitis which do not walk until late, on account either of muscular weakness or of tenderness, may resemble cases of organic nervous disease with true paralysis. The diagnosis must rest on the presence of the general signs of these nervous diseases already described.

The existence of flat-foot in children over two years old should lead to an examination for knock-knee. The combination of these two conditions will in most cases be found to be dependent upon present or previously existing rhachitis.

**PROGNOSIS.**—The prognosis of rhachitis is favorable, provided no complications arise. A spontaneous arrest of the disease may take place in any of its stages, but, as a rule, if the affection is at all pronounced, serious deformities are usually produced. If a hydrocephalic condition, which at times appears in rhachitis, is present to any degree, if there is much diarrhoea, or if the infant is subject to frequent attacks of bronchitis, the prognosis is very unfavorable.

When properly treated, the health of these children improves slowly, and, unless the deformities which have occurred in the bones have advanced too far, more or less complete recovery usually takes place in the third or fourth year.

**TREATMENT.**—The treatment of rhachitis is essentially dietetic and hygienic. The infants should be kept in the open air as much as possible, and should live in rooms accessible to sunlight. The food should be adapted to the age, according to the rules which I have given for the feeding of normal infants during the first two years of life. There does not appear to be any drug which produces a specific effect upon the osseous changes which



Vertical section of leg in a case of infantile scorbutus. The red areas around the femur and tibia represent subperiosteal hemorrhages. (Specimen preserved in the Museum of the College of Physicians and Surgeons, New York.) (Page 1075.)





take place in rhachitis. Phosphorus is considered by some observers to be a valuable adjunct to the general dietetic and hygienic treatment, but, according to our experience at the Children's Hospital, it has not proved to be of any especial benefit.

Where the anæmia is marked, iron in some form should be given, and at times an increase in the fat in the food seems to be beneficial.

There has been much discussion as to whether a form of *acute rhachitis* exists apart from the disease scorbutus, which is now well recognized as occurring in young infants. There are certain cases of rhachitis in which the disease is in the beginning more pronounced and more acute in its development than usual. Again, in the course of an ordinary case of rhachitis acute symptoms may arise. But cases presenting the symptoms to be described under the heading of scorbutus should not be considered necessarily as acute forms of rhachitis on account of the severity of the symptoms, but for the present should be classed as scorbutus supervening on rhachitis.

**SCORBUTUS (Scurvy).**—Scorbutus is a constitutional disease closely associated with imperfect nutrition and having a definite relation to the deprivation of the individual from fresh food. It is characterized by anæmia and a tendency to hemorrhage, and in most cases is accompanied by the condition of the gums which is present in stomatitis ulcerosa.

**ETIOLOGY.**—In addition to the view that the cause of scorbutus is of chemical origin, owing to the significant relation which the disease has to a lack of fresh food, it is supposed that there may be a special micro-organism which causes the disease. This, however, has not been proved, and we have no further knowledge regarding the etiology of scorbutus.

**PATHOLOGY.**—So few post-mortem examinations have as yet been made on infants dying of scorbutus that the pathological lesions have not been finally established. A sufficient number of autopsies, however, has been reported by Barlow and others, notably Northrup, to settle at least the more important features in the pathology of infantile scorbutus.

There are no alterations in the blood, either anatomical, chemical, or bacteriological, which can be considered peculiar to scorbutus. There are deep hemorrhages into the muscles and occasionally about or even into the joints, but the hemorrhage in infantile scorbutus is essentially subperiosteal and chiefly of the long bones. The femora are most commonly affected, and there is a tendency to separation of the epiphyses. There may also be a varying amount of interstitial hemorrhage in the lungs, spleen, kidney, and intestinal glands. Hemorrhages into the mucous surfaces are usually present, the gums being chiefly affected and presenting the condition of stomatitis ulcerosa, which I have described in a previous lecture (page 781).

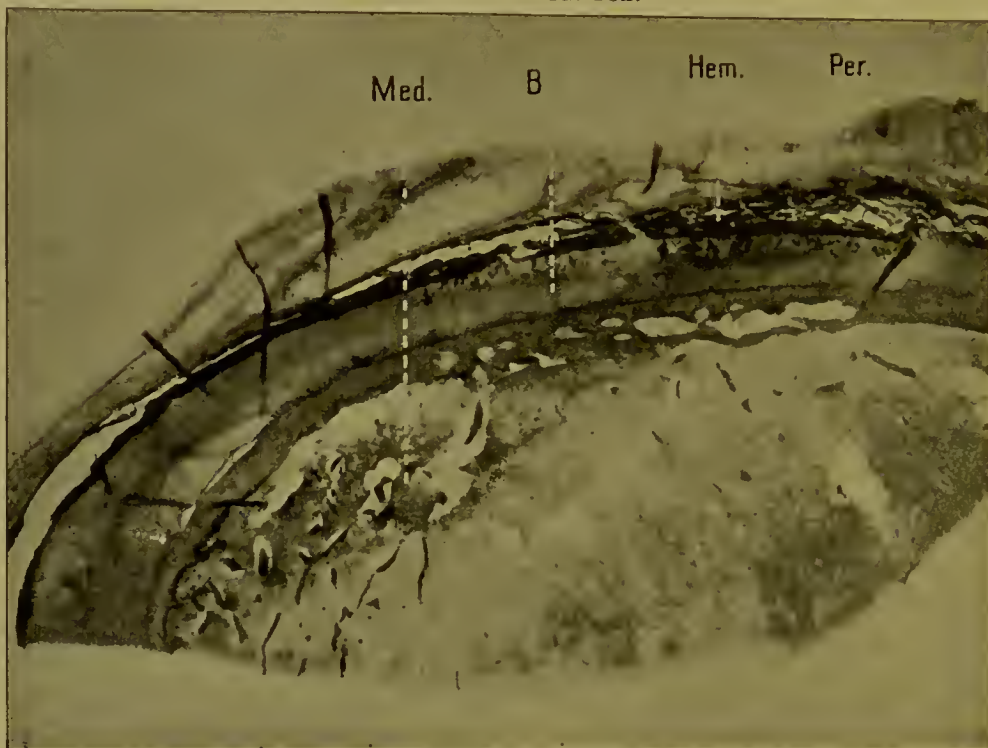
By permission of Dr. Northrup, I have had a section made of the bones of the leg of an infant (Case 518, Fig. 151, facing page 1074) who died of scorbutus, under his care.



On examining this specimen you will see that the femur is normal at its upper extremity. The lower half is invested with a black, grumous, subperiosteal layer of blood. The lower epiphysis is detached, and the lower end of the shaft, macerated, eroded, and soft, is lying loose in the black, disintegrating blood-clot. The tibia is surrounded by thin, dark, hemorrhagic layers beneath the periosteum, and the proximal portions are congested. The fibulae and the bones of the upper extremities were normal.

Here is a microscopic section (Fig. 152, Case 519) of this bone, which shows no syphilitic or rachitic changes in the bone or the periosteum.

CASE 519. FIG. 152.



Section of scorbutic bone. Med., medulla; B., bone; Hem., hemorrhage; Per., periosteum.

The soft macerated bone gave no evidence of suppuration, but there was a moderate congestion of the femur and the upper extremity of the tibia.

**SYMPTOMS.**—The symptoms of infantile scorbutus are those of a slow and progressive caehexia. The infants become anæmic, and show more or less gastro-enteric disturbance of a subacute functional type. Profuse sweating, especially about the head, at times slight feverish attacks, and lessened appetite, are among the early symptoms. The temperature may be from time to time slightly raised, but not significantly so. The first symptom, however, which especially attracts the attention is a sensitive condition of the bones. The infant cries when the affected parts are touched. It does not seem to suffer pain when it is allowed to remain quiet, but as the disease advances the expression of its face indicates the fear of being handled. My individual experience with infantile scorbutus has been derived from sixty or seventy cases, all of which, with few exceptions, were from eight to twelve months of age. I have met with no cases later than the first half of the second year, and with none earlier than the first half of the first year.

As the disease progresses, more marked symptoms develop. Swellings of the limbs, usually of the diaphyses just above the epiphyses, appear.

These swellings are most common and most prominent in the legs, but may also appear in the bones of the forearm. They are usually pyriform and symmetrical in shape, the skin over the swelling being more or less tense, but not fluctuating. There is commonly some tenderness on pressure, but, as a rule, no especial heat of the affected part. The pain and swelling do not seem to be in the joint, but in the diaphysis and epiphysis. Signs of hemorrhage may occur in the skin over the affected parts, appearing at first as small blue maculæ and later involving larger areas, as though a deep hemorrhage were coming to the surface. In advanced cases hemorrhage may take place to such an extent in the deeper parts around the eyes that the eyes will be pushed forward (proptosis).

Where the infant has not cut any teeth, the mucous membrane of the gums, according to my experience, has not been affected; but where a tooth is pressing on the gum and is almost through, or even where a small portion of a tooth has penetrated the gum, small areas of congested mucous membrane appear, and are of great aid in the diagnosis. In some cases a few hemorrhagic maculæ appear in other parts of the skin, as of the forehead.

In addition to the symptoms of epiphyseal pain, the infant keeps the affected limb perfectly still, so that, unless it were understood that it is pain which prevents it from moving the limb, it might be supposed that it was paralysis; in fact, this symptom in scorbutus has been termed pseudo-paralysis. It has, of course, nothing to do with true paralysis, and corresponds to what is seen in rheumatic affections of the joints.

DIAGNOSIS.—The diagnosis of infantile scorbutus is to be made from rheumatism, rachitis, purpura, syphilis, and spinal paralysis.

In the diagnosis from rheumatism the absence of heat and tenderness of the joint and of a pronounced rise of temperature is usually sufficient to distinguish the two diseases.

The diagnosis from rachitis is to be made by the presence of hemorrhages, the intense pain in the region above the epiphyses, the absence of a rachitic rosary, and the absence of symptoms of rachitis when it is not coexistent. If teeth are present, the occurrence of stomatitis ulcerosa usually makes the diagnosis clear. Out of all my cases there have been only a small number, perhaps a dozen, that have shown any symptoms whatever of rachitis. In these cases where rachitis was present the symptoms of scorbutus appeared to complicate a primary rachitis, and when the scorbutic symptoms passed away the rachitic manifestations remained.

Purpura, except in the severe forms in which the joints are affected, is easily differentiated by the absence of the peculiar osseous symptoms of scorbutus.

Scorbutus is differentiated from syphilis by the extreme tenderness, the hemorrhages, and the commonly occurring stomatitis ulcerosa which occur in the former disease, while syphilis has distinctive symptoms which are not found in scorbutus, and which I have already described (page 491).

The differential diagnosis between scorbutus and spinal paralysis is made



by the presence in the former of enlargement and tenderness in the neighborhood of the epiphyses. Pain is present only in the initial stage of spinal paralysis, and tenderness is absent. In spinal paralysis, also, the onset is sudden, and there are no premonitory symptoms.

PROGNOSIS.—Scorbutus is very variable in its duration. If left untreated, all the symptoms may become more pronounced and the infant finally die of exhaustion. When properly treated, and uncomplicated by any other disease, the prognosis is very favorable if treatment is begun early in the attack, before the vitality of the infant has been too much reduced.

TREATMENT.—The treatment of infantile scorbutus is essentially by changing the improper food which in most cases is being given, to fresh milk and orange juice. Under this treatment the pain and tenderness of the limbs rapidly disappear, sometimes within a few days, as does also the stomatitis ulcerosa. In the beginning the juice of one orange should be given in the twenty-four hours. If a rapid improvement does not take place, a still larger dose should be given within a few days. These scorbutic infants usually take orange juice with avidity, but they should be forced to take it if they do not like it. The nurse should be cautioned to move the affected limbs as little as possible, and the infant should be kept on a comfortable pillow on which it can be carried about.

In my earlier cases, before I recognized the scorbutic element in the disease, I treated these infants with a number of drugs, none of which appeared to have the slightest beneficial effect. In some of these cases the symptoms grew progressively worse, and the infants died. In one of them, however, where the hemorrhages in the skin were extensive and where proptosis was marked, the infant recovered entirely when a properly modified fresh milk was substituted for the artificial food which it had been taking. In some of the later cases which I have seen in consultation, where infants living in the country with good hygienic surroundings were being fed on one of the many artificial foods, the disease had progressed to such an extent that the infants were extremely anæmic, had hemorrhages in various parts of the skin, were unable to take any food, and were seemingly dying; in fact, they were as much reduced as were the cases which I have just spoken of as having terminated fatally. These infants, after taking orange juice for a few days, invariably improved rapidly, and usually recovered entirely in two or three weeks.

In my experience there is no evidence that sterilized milk is a cause of scorbutus. If the milk is properly modified it can be heated to 75° C. (167° F.), or even to 100° C. (212° F.), without, so far as I am aware, having a deleterious effect upon the osseous system.

All my cases have presented in different degrees the symptoms which I have just described, and which are well represented in this infant whom I have here to show you to-day.

This infant, a female (Case 520), ten months old, was healthy at birth and weighed 3636 grammes (8 pounds). It was nursed at first, but later was fed on a patent food, on

which it did not gain. When it was eight months old it lost somewhat in weight, had profuse sweating, and began to have tenderness in its limbs. It has six teeth. On looking at the infant you see an expression of fear on its face, and also that it keeps its arms and legs perfectly motionless.

## CASE 520.

## I.



Infantile scorbutus. (Second month of disease.) Female, 10 months old.

Whenever it thinks that I am about to touch the legs or the arms it cries with fear. There is no evidence of rhachitis in this infant. You see that there is a swelling of the diaphysis just above the epiphysis of the bones of the right wrist, and also in the lower part of the femur of each leg and the lower part of the tibia. The swelling does not fluctuate, has a hard, tense feeling, and apparently is not connected with the joints. There is no increased heat of the skin, but there are certain circumscribed areas of hemorrhage in the skin over the swellings. The gums show the condition of stomatitis ulcerosa to so marked a degree that they almost cover the teeth. They are purple, bleed easily, and are very similar to those seen in the case of scorbutus which I showed you at an earlier lecture (Plate VIII., Scorbutus, facing page 781).

## CASE 520.

## II.



Infantile scorbutus. (One month after treatment.) Female, 10 months old.

(Subsequent history.) The infant's diet was changed to a modified milk, and it was given the juice of one orange daily. Within two days it moved its legs and arms freely, the anxious expression left its face, and in a few weeks it had gained much in weight and was perfectly well (II.).

There was no evidence of rhachitis.

An examination of the blood in this case gave the following result:



## BLOOD EXAMINATION 38.

Red corpuscles . . . . .	4,435,000
Hæmoglobin . . . . .	35 per cent.
White corpuscles :	
Small mononuclear . . . . .	8   “
Large mononuclear . . . . .	44   “
Polynuclear leucocytes . . . . .	57   “
Eosinophiles . . . . .	1   “

The blood examinations in two other cases gave the following results :

## BLOOD EXAMINATION 39.

Red corpuscles . . . . .	4,660,000
Hæmoglobin . . . . .	45 per cent.
White corpuscles :	
Small mononuclear . . . . .	5   “
Large mononuclear . . . . .	73   “
Polynuclear leucocytes . . . . .	22   “

## BLOOD EXAMINATION 40.

Red corpuscles . . . . .	4,602,500
Hæmoglobin . . . . .	(not obtained)
White corpuscles :	
Small mononuclear . . . . .	10 per cent.
Large mononuclear . . . . .	68   “
Polynuclear leucocytes . . . . .	21   “
Eosinophiles . . . . .	2   “

**RHEUMATISM.**—Rheumatism is a non-contagious febrile disease, when affecting children usually subacute, and characterized by pain sometimes in the joints and sometimes in the muscles.

**ETIOLOGY.**—The cause of rheumatism is not known, although that the disease is incited by exposure to cold and dampness is evident. It is possible that it is microbic in its origin, this view being strongly supported by the intimate relation between rheumatism and endocarditis, since the latter disease has been proved to be of bacterial origin. Acute articular rheumatism is rare in early life, though it may occur at any age. Subacute attacks of rheumatism, characterized by pains in various parts of the body and limbs and a moderate heightening of the temperature, are very common in childhood. The more severe forms of rheumatism which occur in adults, such as arthritis deformans, are very rare in children. The chronic form of rheumatism is also rare in early life. The chief characteristics of rheumatism in young children are that often it does not involve the joints, and that the milder forms of the disease are much more apt to be complicated by endocarditis than is the case in adult life.

**PATHOLOGY.**—There are no lesions which especially characterize the pathology of rheumatism. The lesions which occur in the course of the disease are those of other diseases, such as endocarditis or pericarditis, which so frequently complicate it. Small subcutaneous fibrous tumors at times appear during an attack of rheumatism, especially in children, and may be

found in any part of the body or limbs. They seem to be closely connected with rheumatism, and the cases in which they occur are frequently associated with endocarditis.

**SYMPTOMS.**—The symptoms of rheumatism when uncomplicated vary according as the disease is the acute articular form or locally affects the muscles of various parts of the body, such as the neck (torticollis); sometimes the disease is simply represented by indefinite pains, which may last for a number of days and then disappear to recur at a later period. The symptoms are, as a rule, not so severe as in later life, even when the joints are affected, and in the few cases of articular rheumatism in children which have come under my care the suffering has been very slight in comparison with what is experienced in adults. In the acute articular form there are swelling, tenderness, and redness of one or more joints, accompanied by a heightened temperature and loss of appetite. A very common accompaniment of rheumatism is anæmia. The disease runs a varying course of three to six weeks, unless complicated by some other disease. The most common complications are endocarditis and pericarditis, and when these diseases appear the symptoms of these complications become prominent. In some cases the endocarditis may appear before the development of the rheumatic symptoms.

**PROGNOSIS.**—The prognosis of rheumatism in children is very favorable, unless complications arise, in which case it depends upon the severity of the complication.

**TREATMENT.**—Rheumatism in the articular form is a self-limited disease, and the treatment is purely symptomatic. The child should be kept in bed in a room of an equable temperature, 20° to 21.1° C. (68° to 70° F.). The affected joints should be wrapped in cotton wool. No applications to the joints are, as a rule, indicated. For the alleviation of the pain salicylate of sodium in moderate doses according to the age of the child is valuable; but there is no drug which is in any sense curative of rheumatism, and salicylate of sodium has not been found to lessen the frequency of cardiac complications. The oil of gaultherium can also be used, and has about the same efficacy as salicylate of sodium. Opiates are seldom needed. A careful physical examination should be made every day in these cases of rheumatism, in order to detect the cardiac complications which are so likely to arise. During the acute stage of the attack the diet should be broths and milk. A number of careful observers believe that an alkali, such as citrate of potassium, should be given in conjunction with the salicylate of sodium.

I have some cases here in the wards which illustrate the different forms of rheumatism in children.

Here is a boy (Case 521, page 1082), three years and four months old, who has been treated in the hospital for bronchitis, and when he was convalescent from that disease was attacked with acute articular rheumatism.

There is no rheumatic history in his family, and he has never had rheumatism nor any other disease except the bronchitis for which he was admitted to the hospital. After having



been feverish for two days, the temperature varying from 37.7° to 38.8° C. (100° to 102° F.), he complained of pain and tenderness in his shoulders, wrists, and elbows. On the following day these symptoms increased, being especially marked in the left hand and left knee. You see the expression of anxiety on his face, showing that he fears that the tender joints will be touched. The weight of the bedclothes is kept from the knee by this cradle,

CASE 521.

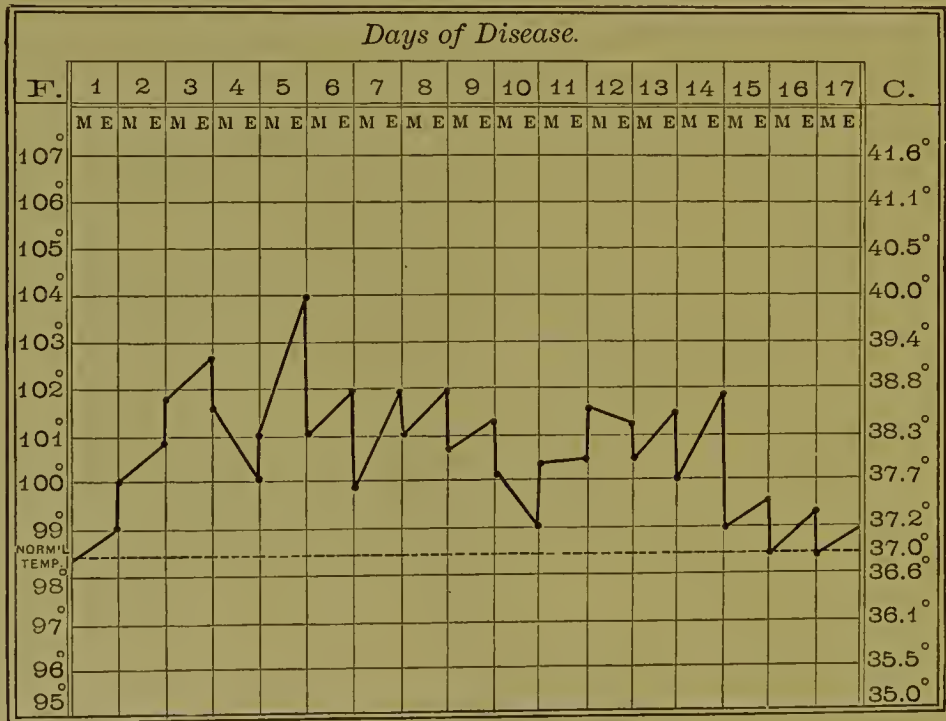


Acute articular rheumatism. Adult type of disease. Male, 3½ years old.

and the arm is comfortably arranged on a pillow. These details in the nursing of a rheumatic child are very important. The cotton wool has been removed from the joints, that you may see how swollen and reddened they are. He is being treated with oil of gaultherium, 4 minims every three hours. The temperature has risen to-day to 39.7° C. (103.5° F.). An examination of the cardiac region does not detect any cardiac complication.

(Subsequent history.) The child suffered considerably for four weeks, but at the end of that time the joints gradually grew less painful, and he was entirely well thirty-three days from the onset of the attack.

CHART 48.

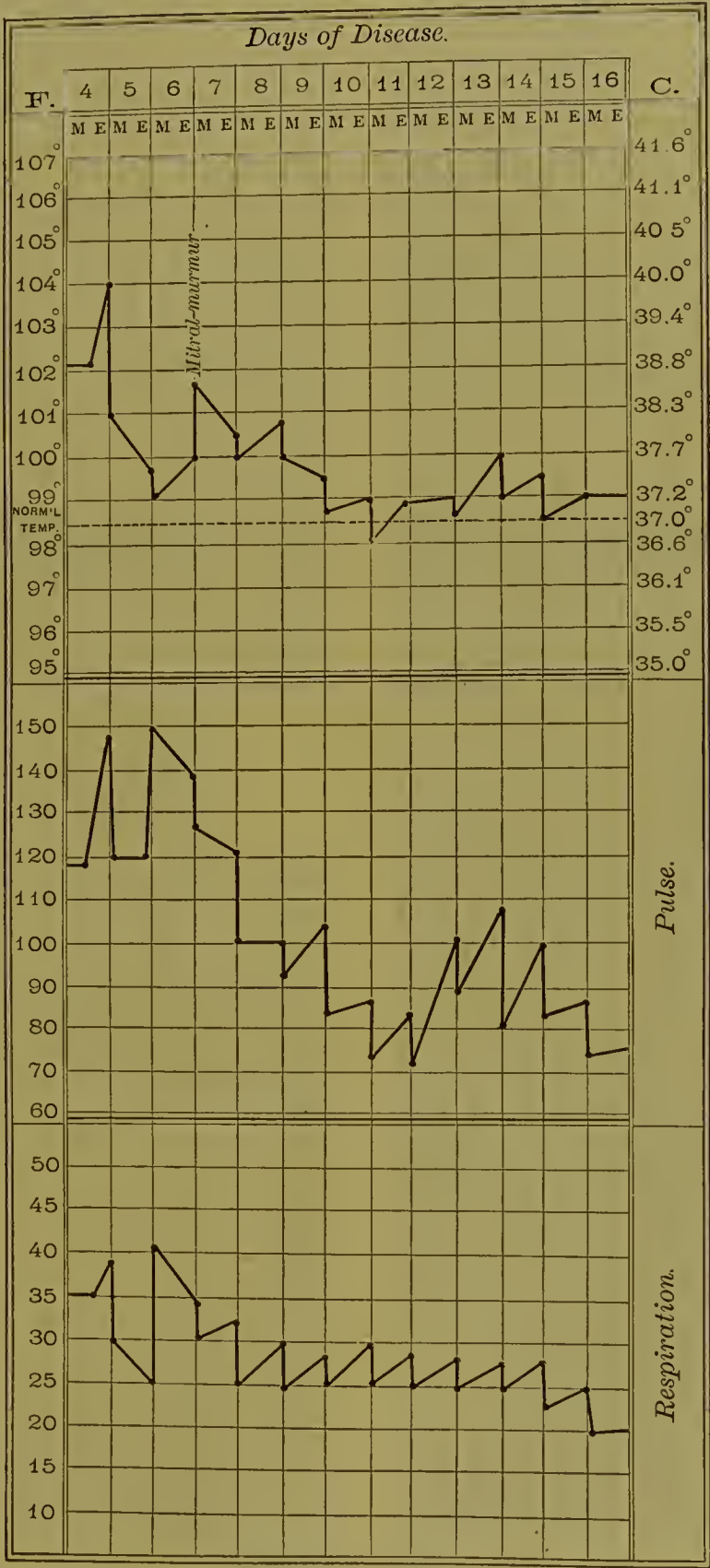


Acute articular rheumatism.

Here is a chart (Chart 48) which shows the range of his temperature for the first seventeen days.

This next child, a girl (Case 522), five and one-half years old, is interesting as illustrating a number of characteristics in connection with the rheumatism of children.

CHART 49.



Acute articular rheumatism. Acute endocarditis on seventh day from beginning of attack.

She was attacked eight days ago with pain, swelling, and tenderness in her left ankle. On entering the hospital her temperature was 40° C. (104° F.), her pulse was 145, and her



respirations were 40. There was very slight pain in the joints, and, although her appetite was lessened, she otherwise seemed well, and she has not complained of any pain since the beginning of the attack. On the sixth day the temperature fell to  $37.2^{\circ}\text{C}$ . ( $99^{\circ}\text{F}$ ). It has been interesting to note the extreme latency of the disease, and how the child has seemed to be perfectly comfortable from the beginning of the attack, except when the ankles, both of which are swollen and tender, were touched. Yesterday, the seventh day of the disease, the temperature rose to  $38.6^{\circ}\text{C}$ . ( $101.5^{\circ}\text{F}$ ), and an examination of the chest showed a mitral systolic murmur. To-day the murmur is more marked, and is transmitted to the axilla and the back. The area of absolute cardiac dulness is slightly increased, and extends to the middle of the sternum.

(Subsequent history.) By the end of the second week of the attack the pain and tenderness had left the ankles, and the child seemed quite well. The area of absolute dulness was found to be normal, but the systolic murmur still continued.

The chart (Chart 49, page 1083) shows the rheumatism gradually subsiding up to the seventh day of the disease, when the endocarditis arose as a complication.

I have here a boy (Case 523) who during an attack of rheumatism developed the subcutaneous fibrous nodules which I have just described.

#### CASE 523.



Rheumatism. Subcutaneous fibrous nodules. Male, 13 years old.

When he was seven years old he had an attack of rheumatism affecting his ankles and the muscles of his neck. His temperature was  $37.2^{\circ}\text{C}$ . ( $99^{\circ}\text{F}$ .); his urine was normal. During this attack a systolic souffle transmitted to the axilla developed, and the area of

CASE 524.

I.



Acute rheumatic torticollis. Fifth day of attack.

CASE 524.

II.



Acute rheumatic torticollis. Sixth day from beginning of attack. Recovery. Male, 5 years old.





absolute cardiac dulness was increased. This attack lasted eight days. He is now thirteen years old, and ever since his rheumatic attack, six years ago, he has had more or less dyspnoea on exertion, and at times cardiac pain, but he has never had any marked return of the rheumatism. He has lately noticed these small lumps appearing under his skin. When they were first noticed he had indefinite pains in his limbs, severe headache, and malaise. Some of the lumps are slightly tender. You see that they are on the chest, arms, abdomen, and legs, mostly on the anterior surface.

I have here an interesting case of the acute localized form of rheumatism which sometimes occurs in children.

This boy (Case 524, facing page 1084) is five years old. Since he was three years old he has been subject to attacks of torticollis, apparently of rheumatic origin. Five days ago he was brought into the hospital in one of these attacks. His temperature is somewhat raised, and he has a slight loss of appetite, but otherwise he is perfectly well, and he does not suffer any pain except when his neck is touched. The head, as you see, is drawn rigidly back. These paroxysmal attacks usually last two or three days, when they ~~pass~~ pass off as suddenly as they came. The last attack which he had was one year ago.

(Subsequent history.) On the following day the stiffness and the pain in the neck passed off, and the head resumed its normal position.

Various drugs have been given in these attacks, but none with any especial benefit except salicylate of sodium, which seems to control the pain.

I have also had under my care a little boy (Case 525), about four years of age, who was attacked with fever, pain in the region of the spine, and spasm along the entire length of the spinal column. There was no pain or tenderness anywhere except over the vertebral column, and these symptoms were not so marked in the cervical region as lower down. The child had no mental disturbance, but for a number of days was in a condition of continued opisthotonos from the hips upward, and he had to be kept in a reclining chair with pillows under his arched back. The normal functions of the bladder and intestine were not interfered with. The pulse was quick, the temperature was moderately raised, and the respirations were normal. The appetite was lessened.

He remained in this condition for about a week, when the spasm of the back began to disappear. The muscles relaxed for a short time and then stiffened again. Finally complete relaxation took place, and the child recovered entirely.

The attack was acute in its onset, and did not follow any injury. The treatment was with bromide of potassium, 0.3 gramme (5 grains) three or four times in the twenty-four hours.

It seems to me that this case can be classed as one of spasmodic rheumatism.

Although acute articular rheumatism is rare in infancy, I have met with a number of cases at this early period of life.

I have already referred to the little girl (Case 41, page 127), two years old, who, after exposure, was attacked with acute rheumatism in both hip-joints.

I have also met with a case of general acute rheumatism attacking all the joints, in an infant (Case 526) two weeks old, after exposure to a cold draught while being bathed. Any movement of the joints caused the infant to scream. He lost rapidly in weight, his surface circulation was disturbed, and the attack lasted for four months; but when he was six months old he was perfectly well, and no cardiac complication developed during the attack.

Another case of this kind was an infant (Case 527) who was attacked with general acute rheumatism when she was seven months old, the attack lasting until she was fifteen months old, when she recovered without any cardiac complication, and who is a well, strong child to-day.



Cases of this kind must, of course, be differentiated from seorbutus, which sometimes closely simulates rheumatism, and of the diagnosis of which I have already spoken.

**ACUTE ARTHRITIS OF INFANTS.**—A disease usually confined to one joint, probably starting as an acute infection of the epiphysis, and followed by an effusion into the joint which rapidly becomes purulent, has been called the *acute arthritis of infants*, and must be distinguished from rheumatism. The disease is essentially surgical in its nature.

The *symptoms* which would lead you to suspect that a more serious affection than rheumatism had attacked the infant are not definite, but are somewhat as follows. The disease occurs only under two years of age, and usually in the first year. The onset is sudden. There are a heightened temperature and intense pain, sometimes in a number of joints, but usually localized in one joint, and accompanied by symptoms of a character grave beyond what would be expected in rheumatism. As in rheumatism, the part affected is swollen, reddened, tender, and fixed, but the surrounding soft parts are also involved to a greater degree than when the attack is of rheumatic origin, and the swelling, at first tense, soon grows fluctuating as the joint becomes involved. From the beginning of the disease the signs of sepsis, as shown by great prostration, rapidly supervene.

The *prognosis* is very unfavorable, unless immediate and radical surgical treatment is carried out; but a number of cases have been cured.

The case should be placed at once in the hands of a surgeon. The *treatment* should be immediate and free incision of the joint.

**PURPURA.**—Purpura is a term applied to certain conditions in which there are hemorrhages into the skin or mucous membranes. These hemorrhages may be of various sizes. When small, they are called petechiæ; when larger, they are called ecchymoses. There is no proof that purpura is a disease of the blood. Its etiology is very obscure, and, although this condition has been divided into various forms, such as purpura simplex and purpura hæmorrhagica, it is doubtful whether these are not all microbic in their origin and simply represent different degrees of infection.

In the more simple forms of purpura the hemorrhages are only in the skin, while in the more severe affection the mucous membranes of the mouth and gastro-enteric tract are usually involved.

Not only does purpura occur in what may be called primary forms, but this purpuric condition may also be secondary to a number of diseases, especially those of an exhausting nature. Thus, I have seen it in the more severe and later stages of infantile atrophy, where the hemorrhages may cover almost the entire front of the body. It may also be a symptom in the more severe cases of measles, scarlet fever, varicella, variola, and diphtheria.

In an infant (Case 528) who died of infantile atrophy at the Infants' Hospital the skin of the extremities showed numerous ecchymoses of various sizes and of a dark red and purple color. On the thorax on both sides above the nipples were two large ecchymoses, and

there were smaller ecchymoses all over the rest of the trunk. On post-mortem examination nothing abnormal was found except a slight atelectasis of the lower lobes of both lungs, with pleuritic adhesions at the base of the right lung and slight granular degeneration of the heart, liver, and kidneys, with hyperplasia of the mesenteric lymph-glands.

In the simple forms of purpura the disease in children is often mild, and is accompanied by a loss of appetite, slight anæmia, a slight degree of fever, and the appearance of petechiæ in various parts of the skin. The prognosis is good, and the duration of these attacks is usually from one to two weeks. They are at times associated with pains located in various places. It is possible for purpura to develop in the course of a rheumatic attack of great severity or where the infant's vitality is much reduced, just as it might appear in any prostrating disease.

The form which has been called *purpura rheumatica* (peliosis rheumatica; Schönlein's disease) probably has no connection with rheumatism beyond the possibility of their both being microbial, and merely simulates rheumatic arthritis from the fact that it affects the joints. The diagnosis is made by the characteristic association of multiple arthritis with purpura and urticaria. Closely simulating and probably representing purpura rheumatica, except that the gastro-enteric symptoms are more prominent, is a form which has been called *Henoch's purpura*. It occurs especially in children between the ages of three and nine years. Its direct cause is not known, although it usually occurs among children who have bad hygienic surroundings and have been ill cared for.

The symptoms are more or less malaise, and pains not especially localized, but chiefly occurring in the extremities and back, sometimes accompanied by slight œdema of the part affected. These early symptoms of pain occur in one or more joints, usually on the outer sides, and sometimes there are swelling and redness simulating articular rheumatism. In this stage there may be a sudden rise of temperature. Accompanying these symptoms there may be a few purpuric spots, but, as a rule, there is a period of several days between the appearance of the pains in the joints and the purpuric appearances on the skin. The purpuric spots may coalesce, and thus form ecchymoses of various sizes and of various colors. They are very apt to begin on the lower leg and spread up to the thighs, genitals, and body. Somewhat later intestinal symptoms develop. While the purpura is spreading there is severe colic, and the pain is very intractable to treatment. The abdomen is retracted and tender. There is obstinate vomiting. The pulse is weak, and the face has an anxious expression. There is more or less diarrhœa, which usually occurs at the end of an attack of colic. The colic and vomiting sometimes last for one or two days. There may be a little blood in the vomitus and in the movements. The vomiting then diminishes, the colic ceases, and later the diarrhœa stops, the pain in the joints passes away, the purpuric spots gradually fade and disappear, and the child, although left in an exhausted condition, is otherwise well.



There are very apt to be relapses, which may appear within a few days or not for several weeks.

These are the symptoms of a typical case ; but there are many variations. As a rule, the younger the child the more typical is the case. Sometimes the purpuric spots closely simulate urticaria. They may occur, although rarely, in the mouth. They sometimes simulate the lesion of erythema nodosum. The attacks of colic have a paroxysmal character. There may be swelling of the joints.

The disease is rarely fatal unless it is complicated by some such disease as nephritis or endocarditis.

The treatment is purely symptomatic.

The most severe form of purpura which occurs is that which is called *purpura hæmorrhagica* (morbus maculosus Werlhofii). The hemorrhages in this form are from the mucous membranes as well as into the skin. The disease begins with debility. A few days later purpuric spots appear on the skin, and subsequently hæmaturia and hæmoptysis occur, from which excessive anæmia may result. There is usually slight fever. When recovery takes place it is gradual, usually occupying two or three weeks.

The prognosis is unfavorable in early life, as death may take place from the exhaustion following loss of blood or from hemorrhage into the brain. The diagnosis of *purpura hæmorrhagica* is to be made from scorbutus by the general history of the case, and by the absence, if teeth are present, of stomatitis ulcerosa.

Very malignant *purpura hæmorrhagica* may occur, sometimes proving fatal within twenty-four hours. This form of purpura is usually spoken of as *purpura fulminans*. It is most commonly met with in infants and in very young children, and is characterized by cutaneous hemorrhages which develop with great rapidity, death sometimes taking place before there has been any hemorrhage from the mucous membranes. I have met with the reports of only seven or eight cases of this malignant form of purpura.

A case of this kind was placed under my care by Dr. W. L. Richardson.

The infant (Case 529) was seven months old, had always been perfectly healthy, and was being nursed by its mother, who was a healthy, strong woman and had a number of other healthy children. The father was also a remarkably strong and healthy man. This infant, without noticeable previous symptoms, suddenly developed this severe form of purpura. Large ecchymoses appeared upon the buttocks and on the trunk, and the infant rapidly failed in strength, and died in twenty-four hours. There was no hemorrhage from the mucous membranes.

**DIABETES MELLITUS.**—In connection with other diseases associated with nutrition I shall mention *diabetes mellitus*, a disease in which sugar accumulates in the blood and is excreted in the urine. The origin of the disease is not known. It is a rare disease in early life.

There does not appear to be any especial difference between the symptoms and course of the disease in children and those which are met with

in adults. A voracious appetite, marked thirst, progressive anæmia, and sometimes emaciation, with the passage of large quantities of urine of a high specific gravity and containing from five to ten per cent. of sugar, are commonly present. Owing to the irritation from the urine, incontinence is quite frequent.

The prognosis is unfavorable, though cases of recovery have been reported. The duration of the disease varies from a few days to a number of months and even years.

The treatment is to reduce the amount of sugar and starch in the food as much as possible. The diet which is most beneficial is milk. I have not found that there is any especial drug which is useful in the treatment of diabetes mellitus. Codeia, from 0.003 to 0.01 gramme ( $\frac{1}{20}$  to  $\frac{1}{6}$  grain) three times daily, has been thought to be useful in reducing the amount of sugar in the urine.

I have met with cases in which there was a transient appearance of sugar in the urine in such diseases as nephritis following scarlet fever. In these instances the sugar disappeared from the urine as the disease in which it occurred improved.

**DIABETES INSIPIDUS.**—Diabetes insipidus is a disease characterized by the passage of large quantities of urine having a low specific gravity and not containing sugar or other abnormal elements.

The etiology and origin of this affection are not known. It is a very rare disease, but is more common in early life than diabetes mellitus, and has been known to be congenital.

Intense thirst, a dry skin, disturbance of the surface circulation, and general nervous symptoms are common in this disease. The children are not apt to show the emaciation which occurs in diabetes mellitus.

Diabetes insipidus is essentially chronic, and so few post-mortem examinations have been made of children dying with this disease that our knowledge concerning it is very limited. There are no drugs which appear to be of benefit in its treatment. The essential part of the treatment is to protect the child from exposure and to see that it is warmly dressed, as sudden changes from heat to cold are liable to increase the general symptoms.

Death usually results from some intercurrent affection. Spontaneous cures have been known to occur.

**TUBERCULOSIS.**—Tuberculosis is a very prevalent affection in early life. While, according to Osler, it is very rare in the new-born, and uncommon in the first three months of life, after this age the number of cases increases very rapidly, and it is very common in the latter part of the first year and in the second year.

It is now supposed that tuberculosis is hereditary in the sense that the infant inherits tissues which are favorable to the development of the disease, except in the rare cases where direct intra-uterine infection has taken place.



I have not used the terms phthisis and consumption, including destructive processes of various organs of the body, because they have been replaced by more definite terms. The terms scrofula and strumous diathesis have not been used, as they are included under the tubercular diseases.

I have spoken of the manner in which the bacillus tuberculosis gains access to the infant's tissues, either by inhalation or in the food. I have also described the manner in which the bacillus tuberculosis affects the various organs, such as the lung, pleura, pericardium, brain, liver, intestine, and lymphatic glands, especially the mesenteric glands (*tabes mesenterica*).

I shall not attempt to describe the various lesions which may occur in tuberculosis when localized, but shall in a few words describe the general tuberculosis which occurs so frequently in infancy.

**ACUTE MILIARY TUBERCULOSIS.**—Acute miliary tuberculosis appears to be more common in the young than in adults. There is always some nidus from which the general infection takes place. The disease occurs as a secondary affection in children who are already tubercular.

After a variable period of loss in weight and general health, which especially occurs in cases where acute miliary tuberculosis is secondary to measles or pertussis, the infant begins to have an irregular type of fever, cough, and general symptoms, such as diarrhoea, capricious appetite, and change of temperament. In some cases the disease advances very rapidly, but often it is of a subacute type, and frequently, unless the tuberculosis markedly affects some organ, such as the lung, the symptoms are very obscure, and cannot be diagnosticated from infantile atrophy, death finally taking place from exhaustion or from the development of some localized tuberculous condition, such as a tubercular meningitis. In my experience, this form, which has been called the *typhoidal type* of the disease, and also the latent forms, are peculiarly difficult to diagnosticate.

**CHRONIC DIFFUSE TUBERCULOSIS.**—Where instead of the miliary lesions which characterize acute miliary tuberculosis a chronic diffuse form of tuberculosis arises, the symptoms are more marked, and usually are so closely connected with the bronchial lymphatics and the lungs that it is more easily diagnosticated. This latter is one of the more common forms of tuberculosis in children.

The prognosis of general tuberculosis in early life is very unfavorable, and there is no known treatment which is of any benefit.

To illustrate how extremely latent and masked may be the symptoms of miliary tuberculosis, I shall show you the results of a post-mortem examination which has just been made on an infant dying of that disease.

This infant (Case 530) was seven months old, and was in the hospital from October until December. During the time that it was in the hospital it became extremely emaciated, diarrhoea occurred from time to time, and there was an irregular and varying temperature, which was never especially high. It had a purulent discharge from the right ear and a serous discharge from the left ear about a week before its death. There were no other symptoms, but it failed rapidly, and died yesterday.

Anatomical Diagnosis.

- Miliary tuberculosis of the pleura, spleen, kidney, and liver.
- Chronic tuberculosis of the bronchial lymph-glands and of the lung.
- Broncho-pneumonia.

Here is an infant (Case 531), one and one-half years old, who was brought to the hospital a few days ago to be treated for an attack of bronchitis. On entrance it was much emaciated, and it has since been rapidly failing. I am able to find no marked signs beyond

CASE 531.



General tuberculosis. Multiple abscess. Infant, 1½ years old.

a subacute bronchitis. There is at times a slight cough, the temperature is moderately raised and of an irregular type, and I suspect that the disease is one of the latent forms of general tuberculosis with a tubercular broncho-pneumonia. On examining the chest, back, and legs, especially the buttocks, you will notice that there are numerous subcutaneous abscesses of various sizes, and there are also a few on the head. These abscesses are probably of a tuberculous nature, and a provisional diagnosis of general tuberculosis, with involvement of the skin and the subcutaneous tissues, can be made.

(Subsequent history.) The temperature in this case had been of an irregular type, and not especially raised until five days before the infant died, when it began to rise, and is as represented in this chart (Chart 50).

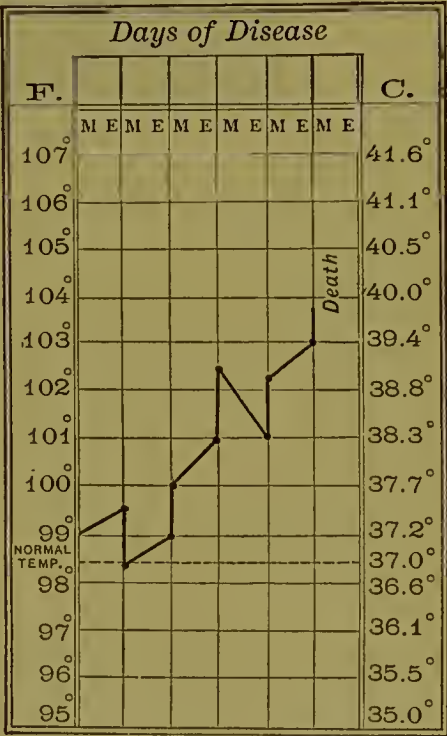
The post-mortem examination, made by Dr. Mallory, showed that there was chronic tuberculosis of the bronchial glands, with acute miliary tuberculosis of the pleura, lungs, spleen, kidneys, liver, and meninges of the brain.

In addition to general tuberculosis, there are certain localized forms of the disease. The more important of these I have already spoken of, but I have here a boy (Case 532, page 1092), nine years old, who, when three years old, had a localized tuberculosis of the little finger (tubercular daetylitis) of his left hand, which has recovered entirely.

I have had him brought here to show you how completely these localized forms of tuberculosis may recover, and I shall call your attention to the cases of tubercular and syphilitic daetylitis which I showed you in a previous lecture (pages 502, 509).

The only other important form of tuberculosis which I have not yet

CHART 50.



Acute miliary tuberculosis.

dwelt upon is localized tuberculosis of the cervical lymph-glands. These I shall speak of in connection with non-tuberculous adenitis.

CASE 532.



Complete recovery from tubercular dactylitis.

**EPIDEMIC INFLUENZA.**—Epidemic influenza is an acute, highly infectious disease, caused by a specific organism which has been described by Pfeiffer.

The period of incubation is short, usually a few hours; relapses are common; one attack does not protect from another.

**SYMPTOMS.**—The symptoms of influenza are very variable. At times they are the same in children as in adults, but in infants and young children the symptoms are often not so severe as in the adult, although they vary in different epidemics, as do those of the adult. It is a characteristic of epidemic influenza that it has no distinct group of symptoms of its own. The symptoms are chiefly a catarrhal affection of the nose and throat, and frequently of the bronchi. These symptoms in young children are accompanied evidently by great discomfort, at times amounting to pain, in the limbs and body, although on account of the age of the patient it is impossible to determine whether much pain is present. Sometimes the only marked symptom is a heightened and irregular temperature, with marked apathy, and the disease may be so slight as to be recognizable only during an epidemic. In older children the symptoms, although, as a rule, not of so severe a type



as in adults, are at times quite serious, especially if continuous vomiting occurs. Severe headache and delirium are present in some cases, and extreme emaciation, out of proportion to the fever or to the morbid conditions detectable on physical examination. Severe symptoms connected with the larynx and the lungs may also arise and rapidly disappear.

**DIAGNOSIS.**—The diagnosis of epidemic influenza is often difficult, unless influenza is present in the community, and is to be made by the careful elimination of other diseases.

**PROGNOSIS.**—The disease in itself is not dangerous, but complications are especially liable to arise and make the prognosis much more serious. These complications are very numerous. They may be meningitis, otitis, ileo-colitis, broncho-pneumonia, and lobar pneumonia. The most common and dangerous complication of influenza is pneumonia, which is usually a broncho-pneumonia, and is of serious import, especially if the child is debilitated at the time of the attack by some previous disease.

**TREATMENT.**—In the treatment of epidemic influenza in infants and children I have found that drugs have very little effect upon the general discomfort caused by the pain. Small doses of phenacetine, 0.06 gramme (1 grain) once in three or four hours, with ten or fifteen drops of brandy, seem to yield as good results as any other mode of treatment. Where there is severe and continuous vomiting, small doses of iced champagne by the mouth and enemata of bromide of potassium, and, if necessary, hydrate of chloral, are indicated. The diet should be milk and beef tea.

During the epidemic of influenza which occurred in Boston in 1891 I had under my care at the Infants' Hospital seven infants, varying in age from a few months to one and a half years, all of whom had epidemic influenza. The symptoms were such as I have described. The infants cried continuously, the temperature was slightly raised,  $37.7^{\circ}$  to  $38.3^{\circ}$  C. ( $100^{\circ}$  to  $101^{\circ}$  F.), and the duration of the attack was about one week. Pneumonia occurred in two of the cases, and in both of these the infants died. Here are the charts (Charts 51 and 52, page 1094) showing the temperature in these cases during the course of the influenza, and the rise when the infants were attacked with pneumonia.

I have in my notes the report of another case, where an attack of influenza was complicated on the eleventh day of the disease by a lobar pneumonia.

The infant (Case 533), sixteen months old, was attacked with catarrhal symptoms of the nose and throat, a slight cough, and a temperature of  $40.5^{\circ}$  C. ( $105^{\circ}$  F.). The respirations were only slightly increased; the pulse was rapid. Nothing abnormal was found on physical examination. The infant was very fretful, had no appetite, cried incessantly, and seemed to have considerable discomfort. On the ninth day from the onset of the attack the temperature fell to  $39.1^{\circ}$  C. ( $102.5^{\circ}$  F.), and on the following day to  $38.6^{\circ}$  C. ( $101.5^{\circ}$  F.). On the evening of this day the infant, who had begun to be brighter and to notice its playthings, again seemed very sick. Its respirations increased in frequency, there was motion of the *alæ nasi*, and the temperature rose to  $40.8^{\circ}$  C. ( $105.4^{\circ}$  F.). On the following day the temperature fell in the morning, but began to rise in the evening, and by the





(102° F.); on the following day it rose to 39.7° C. (103.5° F.) in the evening, and in the next two days gradually fell to 37.2° C. (99° F.). On the following day it rose to 38.6° C. (101.5° F.), and in the next forty-eight hours fell gradually to 36.6° C. (98° F.). At this time the dulness began to disappear, moist râles appeared, the infant became much better, and in a few days, although very weak, seemed bright and well, and the physical signs in the lung had entirely disappeared.

Here is the chart (Chart 53), which shows the temperature during ten days of the influenza, when a lobar pneumonia appeared and ran a course of five days, after which the temperature gradually fell to the normal point. It is possible that this case was one of pneumonia from the beginning of the attack, but it showed all the characteristic symptoms of epidemic influenza, and no dulness was found in the lung until the infant had apparently recovered from its influenza.

**DISEASES OF THE THYROID GLAND.**—The thyroid gland is a highly vascular organ. It covers the front and sides of the upper part of the trachea, and also extends up onto the larynx. Its function is not known. I shall not describe such diseases of the thyroid gland as exophthalmic goitre, which are very rare in early life, but shall refer only to those morbid conditions which you are most likely to meet with,—namely, hyperæmia, inflammation, hypertrophy, and complete absence. Absence of the thyroid gland, disturbance of its function, or actual disease of its tissues, are usually accompanied by peculiar symptoms.

**HYPERÆMIA.**—A temporary congestion of the rich vascular tissue of the thyroid gland occurs under various conditions, such as the approach of puberty. This condition is usually so transient as to be scarcely noticeable so far as the symptoms are concerned, but sometimes it is sufficient to cause dyspnoea from pressure. This usually trivial condition has been thought, however, in certain cases to lead to the production of one of the forms of goitre.

CASE 534.



Hyperæmia of the thyroid gland. Female, 13 years old.

This girl (Case 534), thirteen years old, was first noticed to have a swelling of the thyroid gland two or three weeks ago. The swelling is becoming more prominent. The cata-



menia have not appeared. The girl is well and strong, but is somewhat more fretful and capricious than appears to be in accordance with her usual temperament. The tumor is elastic, does not fluctuate, and is not red or tender. She seems to represent one of that class of cases in which continued hyperæmia of the thyroid vessels occurring at puberty leads to enlargement of the gland. It is possible that a spontaneous lessening in the size of the tumor may take place when the catamenia have been established, but we know so little about this class of cases that the prognosis as to complete recovery must be very guarded. The treatment of this case will be the external application of iodine.

**THYROIDITIS.**—Acute inflammation of the thyroid gland is not very common (Delafield and Prudden), but may occur from a variety of causes. It may result in the formation of abscesses of various sizes or in the production of new connective tissue. According to Osler, acute thyroiditis is rarely primary, being commonly a metastatic affection occurring in the course of some febrile disorder. It has been noticed among children as a complication of measles, and the process in a number of these recorded cases, instead of retrograding spontaneously as it did in others, caused an inflammatory condition in which abscess-formation occurred. On opening the abscesses the pus was found to contain numerous micrococci.

The symptoms of acute thyroiditis are swelling and redness of the gland.

The treatment is essentially expectant, but some previously intractable cases seem to have been benefited by the application of iodine. The patient should be carefully watched, and, if there are indications that suppuration has taken place, an incision should be made at once, as recovery then usually occurs quite quickly.

**ENLARGEMENT OF THE THYROID GLAND (Goitre; Bronchocele).**—Enlargement of the thyroid gland is commonly called goitre. True goitre consists in the enlargement of the old and the formation of new alveoli in the cells of which a greater or less amount of colloid degeneration takes place. The colloid abnormalities of goitre are rarely present in children (Rex), in whom the thyroid enlargement seems to be little more than a continuation of the natural growth, and a true hypertrophy or an excessive development of normal tissue.

Infants have been born with an enlarged thyroid.

**MYXŒDEMA.**—Myxœdema is a constitutional affection characterized clinically by a myxœdematous condition of the subcutaneous tissues and by mental failure, caused by a disturbance of the function of the thyroid gland.

There are certain general symptoms which accompany disturbance of the thyroid function, whether from entire or partial absence of the gland, or from disease of its tissues, such as atrophy. These symptoms are hebetude, with a general thickening of the tissues, and in young individuals a great lack of development, both mental and physical. It is probable that it is a difference in degree or in kind of thyroid disturbance which produces the other symptoms so characteristic of myxœdema. These symptoms I shall presently describe to you in connection with the especial cases.

These various disturbances of the thyroid function may be endemic or sporadic. The endemic cases are represented for the most part by symptoms peculiar to disturbance of the thyroid function, and also, where goitre is present, by symptoms of mechanical pressure. Cases of goitre without cretinism may, however, occur sporadically, and the sporadic cretin, as a rule, has no goitre. Atrophy of the thyroid gland may or may not be accompanied by goitre. I shall not speak further concerning endemic cretinism, which occurs in certain localities, such as portions of Switzerland, and is dependent apparently on some unknown endemic cause which is also liable to produce goitre. As a race, cretins are distinguished by their stunted stature, large, deformed heads, sickly-looking countenances, coarse and prominent lips and eyelids, wrinkled and pendulous skin, loose and flabby muscles, and imperfect mental development, to which are often added goitres of all sizes.

In certain individuals there is a congenital absence of the thyroid gland. This is a condition found in sporadic cretinism, in which the function of the thyroid gland is lost, just as its function is disturbed in goitre and in atrophy of the gland.

Where the thyroid gland has been removed surgically there is at times a condition similar to that which is met with in myxœdema. This condition has been called by Horsley *cachexia strumipriva*.

The head in sporadic cretinism is usually brachycephalic; that is, it is contracted in its antero-posterior diameter and increased in its transverse diameter. Virchow was the first to observe that in these cases there is a premature ossification of the spheno-basilar bone. The sphenoid and the basi-occipital bones should remain separate until about the fifteenth year, and their early ossification explains, according to Virchow, the changes which take place in the form of the cretin skull and face. The characteristics of the cretin bone are an enormous overgrowth of cartilage, an arrest of growth at the distal ends of the bones, and a premature ossification of the shaft. Here is the tibia (Fig. 148, II., page 1066) of a cretin child. The section was made by Dr. Whitney, and is distinguished, as you see, anatomically by the almost entire absence of the zone of proliferation. This narrow line (Z. P.) marks the boundary between the broad area of cartilage above and the prematurely ossified bone of the shaft below.

This little girl (Case 535, page 1098), whom I have had brought to the hospital to show you, and who is just able to stand, and looks as though she were about one and a half years old, seems to be a case of myxœdema.

She is five and a half years old. Her parents were healthy Americans, not blood relations, and did not have goitre. She was born after a severe labor: it was a head presentation, and no instruments were used. Nothing especially abnormal was noticed about her until the twelfth month, when she did not seem so bright as is usual at that age. When four years old she was brought to the hospital. She could not speak, and her mental condition was much enfeebled. She had never had any convulsions, but had always had incontinence of urine and of feces. When seen a year later she appeared to be in good general condition, but her muscles were large and flabby and she had not improved mentally. The circumference of her head is 46.5 cm. (18½ inches). The measurement



from the occiput to the root of the nose is 34.4 cm. ( $13\frac{1}{2}$  inches), across the head from external meatus to external meatus 29.3 cm. ( $11\frac{1}{2}$  inches). The circumference of the thorax is 40.3 cm. ( $15\frac{3}{4}$  inches). There are no irregularities about her head. The forehead is overhanging, and this is rendered more striking on account of the sunken bridge of the nose. The lips are thick, and the tongue, which seems enlarged, is protruded between them. The hearing is said to be good, and the sight is good. She has been able to sit alone since she was one year old, but can stand only with support, and cannot walk. She is bow-legged, and the bones are somewhat enlarged about the epiphyses. The hands and feet are large and puffy, but do not pit. The feet are bright red, the hands less so. The trunk is stout and thick; the spine is straight; the lungs and heart are normal, and I can detect nothing abnormal about the abdomen except an umbilical hernia. The tendon reflexes are normal. Sensation is good. The thyroid gland is not felt. The teeth are good. There

CASE 535.

Myxedema. Female,  $5\frac{1}{2}$  years old.

is a general condition of infiltration of the skin like myxedema. Hebetude is marked. The treatment of this child will be with an extract made from the thyroid gland of a sheep, as this seems to be the only means which we at present know of by which a certain number of these cases are benefited. I have not treated a sufficient number of cases personally to judge whether the thyroid treatment will eventually prove successful. Other observers, however, claim to have obtained decided improvement, both physical and mental, by its use. I shall order .06 c.c. (1 minim) of the thyroid extract at first three times daily, and gradually increase 1 minim every two days until the rectal temperature rises above  $37.7^{\circ}$  C. ( $100^{\circ}$  F.). If during the course of the treatment the temperature should rise over  $37.7^{\circ}$  C. ( $100^{\circ}$  F.), the extract should be omitted for a day or two. In this way you can determine the proper dose for the especial case. I shall also warn the parents that the child must be kept warm, and be removed to a southern climate in the colder months.

I have under my care a little girl (Case 536), two years old, who appeared to be perfectly well and strong during her first year, but did not learn to sit or creep until the last few months, and who cannot stand alone or walk. There are no signs of rhachitis about the child, except that the anterior fontanelle is not closed. Nothing abnormal is found on physical examination, except that the tissues of the hands and feet are thickened and the skin is dry and cold, with at times a bluish tint. She has always held her mouth open and protruded her tongue, which seems to be thicker than normal. She is phlegmatic, and does not care to play. The bowels are constipated. There is no enlargement of the thyroid. Her case seems to be one of myxœdema.

She has been under treatment two months with the extract of the thymus gland. During this period she has grown much brighter mentally, and now creeps about more, wishes to play, and takes more interest than formerly. The tongue is not so much enlarged as before the treatment was begun, and the bowels are no longer constipated. Several times during the course of the treatment the thyroid extract has had to be omitted, as it seemed to cause digestive disturbance with a rise of temperature,  $37.7^{\circ}$  or  $38.3^{\circ}$  C. ( $100^{\circ}$  or  $101^{\circ}$  F.). Omitting the thyroid extract for twenty-four hours, these symptoms would pass away, and it could then be given again.

This case (Case 537) is one of great interest, as it represents so typically the mental and physical characteristics of sporadic cretinism. I am enabled to show it to you through the courtesy of Professor Northrup, who gives the following account of it:

CASE 537.



Myxœdema. Female, 9 years old. Slight improvement after eighty days' treatment with thyroid extract.

"The parents of the child were healthy Americans from Western Pennsylvania, and they were not consanguineous. The father was 45 years old; the mother was 39 years of age, had had several miscarriages and four healthy children, two of whom had died of some acute disease. This little girl, who is now nine years old, is the fifth child. The mother first noticed that the child could not sit up when it was nine months old, that it practically ceased to grow, and now at nine years it is mentally no older than it was at nine months, and physically it has merely thickened. The first impression one gets on looking at the child



is that it is an idiot. Its hands are large and broad. Its color is peculiarly sallow. The hair is thin, long, dry, and without lustre. The eyebrows are present, and are not remarkable in any way. She has the characteristic flattening of the bridge of the nose, diffuse swelling of the under lid and puffiness of the upper lid, and pendulous cheeks. She has thick, pale lips, with a protruding tongue, which is swollen and pale. The lips and tongue have a tendency to dryness. There are fourteen teeth, all of them of the first set. Those in the upper row are eroded, and appear only at the bottom of a series of ulcers in the upper gums. The lower teeth are in nearly the same condition, and the gums are suppurating. An offensive odor is always present in the mouth. The arms, legs, feet, and hands are unnaturally thick. The abdomen is prominent, and there is, as you see, an umbilical hernia. The hand which is resting on its mother's black glove shows the dry, wrinkled condition so characteristic of myxœdema. Perspiration is absent. The skin is pale, and has a peculiar mottled appearance. The soles of the feet and the palms of the hands are dry. There is marked lordosis. The surface of the child does not suggest the feeling of œdema, nor does it pit. The feeling is that of puffiness and flabbiness. The child cannot sit alone. It can, however, stand when once balanced and allowed to grasp some fixed object. The supraclavicular 'pad' of tissue so commonly found in these cases is present. The thyroid gland seems to be present, and is possibly enlarged. Hebetude is shown to a marked degree, and the delayed cerebration is very evident, although the child never speaks except to say, with infinite slowness, 'da—da.'

"(Subsequent history.) The rectal temperature four days before treatment was begun was 36.4° C. (97.5° F.) in the morning, and 37.5° C. (99.5° F.) in the evening. The child was treated with the thyroid extract prepared so that each drachm represented one thyroid gland of a yearling. Of this preparation 0.06 c.c. (1 minim) was given three times a day until the fourth day, when the temperature rose above 37.7° C. (100° F.), and the treatment was stopped for a day. At this time the appetite had improved, and the breath was not so offensive. Two days later the treatment was begun again, and on the eighth day the tongue was found to be considerably smaller. During the next week the temperature remained under 37.7° C. (100° F.). It then rose above 37.7° C. (100° F.), and the treatment was suspended. The first tooth, a canine, was cut at this time. The largest dose which was given during the treatment was 0.24 c.c. (4 minims) three times a day. The child was treated eighty days in this way. The improvement was very slight, but the countenance was brighter, the tongue became much smaller, and the skin less dry. She lost somewhat in weight while under treatment. The constipation, which was marked when the treatment was begun, disappeared, and she was willing to take a much greater variety of food."

Through the kindness of Professor Osler I am enabled to show you this little girl (Case 538), who is four years old.

The parents were healthy, and there was no hereditary taint on either side of the family, none of whom have had goitre. She was the second child: the labor was easy, and she thrived well. She has never had any diseases. Nothing especial was noticed about the child until its second year, when it was observed that she did not attempt to walk or talk, and that she seemed unnaturally quiet and dull. She did not cut her first teeth until she was two years old. In her third year her skin became very pale and waxy, and her face and limbs seemed puffy and swollen. She had developed very little mentally, and could say only one or two words. The other symptoms indicative of a disturbance of the function of the thyroid gland gradually appeared, such as the myxœdematous condition of the subcutaneous tissues and the development of the supraclavicular pad. The thyroid gland could not be felt. The examination of the blood showed a moderate increase of leucocytes and some irregularity in the size of the erythrocytes. When three and a half years old she was 75 cm. (29 $\frac{3}{8}$  inches) tall, and her head measured 52.3 cm. (20 $\frac{1}{2}$  inches). She had been under treatment with tonics for a year, and was reported to take more notice and to look more intelligent. She was then treated with the thyroid extract, and has improved markedly in both her mental and her physical condition. The tongue, which had been thick and pro-

truding, is fast recovering its normal size. She can walk and talk a little, and Dr. Osler thinks that she may be considered to represent a case in which the thyroid extract has produced decided improvement.

CASE 538.



Myxoedema. Female, 4 years old. Marked improvement under thyroid treatment.

**DISEASES OF THE CERVICAL LYMPH-GLANDS.**—The chain of lymphatics in the neck is so closely connected with the lymphatics of the mouth and throat that infection frequently takes place. I have already spoken of the enlargement of the cervical glands secondary to absorption in cases of diphtheria. Localized enlargement of the cervical glands occurs also in tuberculosis, and sometimes is the only manifestation of that disease. The cervical glands may also be enlarged in lymphatic leucæmia and in multiple sarcoma.

Here is a little girl (Case 539, page 1102) who has, in addition to marked chronic tuberculosis of the lungs, enlargement of the cervical glands, which is very probably of tubercular origin.

When the tubercular disease is advanced in other organs there is seldom much benefit to be derived from the treatment of these glands. I show you this case more for the purpose of comparison with some other cases having enlarged cervical glands than for anything of especial interest in connection with this class of advanced tubercular cases.

The cervical glands may be enlarged from a number of causes, as well as from direct infection through the throat. Any irritation of the scalp, ears, eyes, nose, throat, gums, or teeth may cause a temporary or permanent



enlargement of the cervical glands. In some few cases they enlarge without apparent cause, except that the children are anæmic or debilitated. In these cases they often run a rather acute course, and may subside without suppuration having taken place. In other cases suppuration quickly takes place, and in these, as well as where a number of these glands enlarge and coalesce, and where the enlargement lasts for long periods, we should always suspect that the bacillus tuberculosis is present in the glands.

CASE 539.



Chronic pulmonary tuberculosis, with involvement of the cervical lymph-glands.

The first effort in undertaking the treatment of these cases should be to seek for and remove the peripheral source of irritation which exists in most cases. Decayed teeth should be extracted, eczema of the scalp should be treated, and in all cases as much as possible should be done to diminish any irritation in the area of surface drained by the cervical lymphatics. During the active stage of cervical adenitis it is better not to make any application to the glands, but to treat any general disturbances, such as anæmia or debility, which may be present. When the active process has subsided and a chronic condition is left, the glands may become quiescent or may go on to suppuration. In the treatment of this chronic condition you should take into consideration the possibility that the glands may eventually suppurate and leave unsightly scars. As a rule, it is better to have these cases placed in the hands of a surgeon and the glands removed, as there are no especial contra-indications to the operation, and the scar left when the operation is skilfully performed is slight.

I have here a little girl (Case 540) in whom the cervical lymphatics are enlarged to such an extent that they have become a deformity.

Nothing else abnormal nor any other symptom of tuberculosis can be discovered about

## CASE 540.

I.



Chronic cervical adenitis.

II.



Chronic cervical adenitis (after treatment).

the child. These glands should have been removed before they reached such a size as this, as now on their removal a considerable scar will be left.

(Subsequent history.) The glands were removed, and the picture shows what good results can be obtained in these cases by surgical interference.

## CASE 541.



Enlargement of submaxillary glands.

The submaxillary glands are enlarged in children from various causes, but sometimes from no discoverable cause. At times the enlargement of the glands is accompanied by pain and tenderness, constituting a disease



which has been called submaxillary mumps. In the beginning, however, we should not at once make this diagnosis, as the glands may become enlarged and tender from various causes which have no connection with the specific disease mumps.

Here is a little girl (Case 541, page 1103), two years and four months old, who is an illustration of this class of cases. She was suddenly attacked yesterday with a swelling of the submaxillary glands, accompanied by pain and a slight amount of tenderness over the swollen region. She has a history of exposure to parotitis. To-day the swelling has extended under the entire chin and up the left side of the neck to the face and ear.

The diagnosis in a case of this kind must be held in abeyance for a few days, and strict isolation should be enforced, as if the cause of the glandular enlargement proves to be infectious other children should be protected.

(Subsequent history.) The swelling, pain, and tenderness lasted for a number of days and then gradually subsided. Nothing more definite was discovered regarding the case.

**PAROTITIS (Mumps).**—Parotitis is a highly infectious disease which attacks the parotid gland. Its period of incubation is from two to three weeks. The onset of the attack is usually accompanied by a sense of chilliness, a rise of temperature, and a sensation of stiffness and tenderness about the jaws. This is succeeded by a swelling in the region of the parotid gland, which becomes enlarged and tender, rendering deglutition difficult and often very painful. The disease begins on one side, but the other gland is usually involved in a day or two. As I have just stated, the infection is sometimes confined to the submaxillary glands on one or both sides.

The duration of an attack of parotitis is from a few days to a week, but the infection may last for two or three weeks, and it has been stated in some cases to antedate the appearance of the glandular enlargement. In boys at the age of puberty the complication of orchitis at times arises.

Although the symptoms of parotitis are commonly very mild, unusual cases sometimes occur in which the children are quite sick, and there have been cases in which the orchitis was of so high a grade that acute delirium supervened, and in one case reported by Dukes the boy fainted when the orchitis began.

It is sometimes difficult to differentiate parotitis from a simple non-infectious enlargement of the parotid gland or of the glands in its neighborhood. When the parotid gland is enlarged it usually shows a characteristic swelling under and behind the lobe of the ear, so that the lobe is pushed somewhat upward and forward. This swelling increases rapidly, is very tender, is not especially reddened, does not fluctuate, and is accompanied by constitutional symptoms. The diagnosis is readily made if after a few days the unilateral swelling is followed by corresponding symptoms on the opposite side.

Here is a boy (Case 542, page 1105), twelve years old, who was attacked five days ago with swelling and tenderness in the region of the parotid gland, followed in a short time by stiffness and pain at the angle of the jaw, and accompanied by symptoms of loss of appetite and a heightened temperature.

Two days later the parotid of the opposite side was involved. You will notice how

the neck in the part which corresponds to the position of the parotid is swollen on both sides, and how the characteristic swelling which pushes the ear upward and forward is seen on looking at the child from behind.

CASE 542.



Parotitis. Male, 12 years old.

There is no especial treatment for the disease, as it is self-limited and runs a definite course. The children should be carefully isolated, in order that there may be no further spread of the infection. As deglutition is painful, their diet is usually milk and soups. They should be carefully protected from exposure, and should be confined to their rooms. Older children should be confined to bed, as orchitis in boys and trouble with the mammæ in adolescent girls are less likely to arise under these conditions. It is usually better to apply some soft cotton wool to the painful swelling, and to protect it from any irritation.

**DISEASES OF THE EAR.**—We have, gentlemen, studied the normal infant at birth, and have followed it through its various stages of development into childhood, up to the age of puberty. I have also endeavored to make you familiar with the various morbid conditions which are most likely to arise during these early periods of life.

Before closing this course of lectures, however, I wish again to call your attention to the great importance of bearing in mind, in examining infants and children, the common occurrence of some morbid process in the ear. In many cases where the more pronounced aural symptoms are not evident, symptoms which appear obscure, but really are due to some latent disturbance in the neighborhood of the ear, reflex or otherwise, are readily explained when in addition to the presence of some other disease the unusual symptoms are found to arise from the aural complication. The question of diseases of the ear in infancy and childhood has not received from the general practitioner, nor indeed from those who devote themselves especially to children, the attention that it deserves. Even leaving out of



consideration the cases of disease of the middle ear incident to the exanthemata, which I have already dwelt upon (page 558) when speaking of these diseases, serious implications of the ear from other causes are not uncommon during the first year of life.

Von Tröltsch found on examining forty-seven petrous bones taken from twenty-four unselected children that the middle ear was normal in only eighteen. The other twenty-nine ears showed in varying degrees the appearance of a purulent and sometimes, though rarely, of a mucous catarrh. Of the fifteen children with exudation in the middle ear, the youngest was three days and the oldest one year old; five were in their first month, two each in their second and fourth, three in their third, and one each in their seventh, eighth, and twelfth months.

In every five examinations of the ears of new-born children Schwartz found the tympanum filled with pus in two.

Wreden found in eighty ears of children a normal middle ear in only fourteen; purulent catarrh existed in thirty-six, and simple mucous catarrh in thirty; the youngest child had lived twelve hours, the oldest fourteen months. The majority of these cases were, however, from three to fourteen days old.

Edward Hoffman examined twenty-four petrous bones in infants varying in age from thirty-two hours to four weeks, and found the tympanum filled with pus in seven cases.

Of two hundred and thirty carefully examined cases under seven months of age Kutcharianz found the tympanic mucous membrane normal in thirty only. In fifty it showed either slight or intense catarrhal inflammation, and in one hundred and fifty the tympana were filled with pus.

These statements, quoted from Von Tröltsch, serve to emphasize the statement of that author that even from the beginning of extra-uterine life "there is an unusually strong disposition to disease of the middle ear, owing on the one hand to the double influence of the peculiar morphological relations of the ear and the pharynx, and on the other hand to the diseases and conditions of life to which the child is frequently exposed."

We should therefore consider carefully the ear in all cases where the symptoms are obscure, as well as where those diseases are present in which it is well known that aural complications are liable to arise.

The late Dr. Edward H. Clark made a statement, which has since been largely quoted by other writers on otology, to the effect that the physician who neglected the examination of the ear in the course of the exanthemata of childhood might be denominated an unscrupulous practitioner. The statistics which I have just given you show that not only the possible implication of the ear in scarlet fever and measles, but also the inflammation in the tympanic cavity consequent upon acute catarrhal inflammations of the nose and naso-pharynx, as well as the reflex disturbances, should receive your closest attention.

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